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ANNALS
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XXIV

# OTITIS EXTERNA

## BACTERIOLOGICAL AND MYCOLOGICAL STUDIES

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There has been in recent years a new appreciation of the importance of otitis externa as a disease entity. It occurred with disturbing frequency among troops in the South Pacific. According to Gordon,[1] on Guam it ranked second among illnesses causing loss of time from duty. Nelson[2] reported that 50 and sometimes as high as 70 per cent of the cases seen in the practice of otolaryngology in the South Pacific were otitis externa. Its frequent occurrence in civilian practice during hot, humid summer months is emphasized by observers in widely scattered localities. Otitis externa is an acknowledged problem of military and civilian medicine.

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There has been an added stimulus for obtaining a more exact knowledge of this condition. New therapeutic measures and simple in vitro sensitivity tests are now available. These offer the hope of specific and more effective therapy, if etiological diagnosis can be established and if the responsible organisms respond satisfactorily to available therapeutic agents.

During the summer and fall of 1951 we examined bacteriologically and mycologically 1,377 cultures of the normal and 646 of the diseased external ear. The latter came from 405 young adult male patients with otitis externa, 311 unilateral and 94 bilateral. Of the cultures, 499 were taken from one or both infected ears on the patient's initial visit to the dispensary, and 147 were repeat examinations. The data on these were analyzed separately but as there was not significant difference in findings on the initial and repeat cultures, the combined findings are given.

The in vitro sensitivity to seven therapeutic agents was determined on 3,071 bacteria isolated.

## CULTURE PROCEDURES

The culture media employed, except as indicated, were prepared from dehydrated products (Difco) according to the manufacturer's recommendations. Those used included fluid thioglycollate medium (tubed in 16 x 125 mm tubes) eosin-methylene blue agar, blood agar (Bacto-Proteus No. 3 agar plus 5 per cent whole citrated human blood) chocolate agar, Sabouraud's dextrose agar, plain and with penicillin 20 units per milliliter and streptomycin 40 units per milliliter added, and potato dextrose agar. Czapek's medium and corn meal agar, plain and with 1 per cent dextrose added, were prepared in our laboratory. The Sabouraud's media were dispensed in 7-inch tubes to provide a large slant.

Specimens for study were obtained from military personnel at the MacDill Air Force Base, Tampa, and the U. S. Naval Air Technical Training Center, Jacksonville, Florida. All cultures from the normal ear were taken by one of the participating bacteriologists, but some of the specimens from infected ears were taken by previously instructed corspmen on duty in the dispensaries. For obtaining specimens, small cotton-tipped swabs were prepared in the laboratory and sterilized in stoppered tubes (16 x 175 mm). These and the thioglycollate medium were taken to the dispensary. All cultures were collected in duplicate. Each swab was inserted into the external auditory canal—avoiding, so far as possible, contam-

ination from surrounding surfaces. It was gently rotated. That for bacteriological examination was placed directly into the tube of thioglycollate broth; the one for mycological examination, into a dry sterile test tube. In general the infected ears had seropurulent exudate which provided a heavier inoculum than that obtained from the dry normal ear. In the laboratory the bacteriological and mycological studies were conducted in separate divisions.

For the bacteriological studies the thioglycollate broth was incubated at 37.5° C. for 18 to 24 hours for cultures from normal ears and for 6 to 8 hours for cultures from infected ears. Those sufficiently turbid to indicate growth were examined at that time; those remaining clear were incubated up to 48 hours. The broth culture from normal ears was first observed microscopically. If gram-positive, sporeforming rods only were present, no further identification was attempted. If gram-positive coci were found, the culture was streaked to a blood agar plate, and if gram-negative rods were observed, an eosin-methylene blue agar plate was also inoculated. Most cultures from infected ears had relatively heavy growth. The microscopic screening was omitted since it was found that virtually all required plating to both blood agar and eosin-methylene blue agar. No culture was discarded as negative prior to 48 hours of incubation.

The swab for mycological examination was used for the inoculation of one slant of plain Sabouraud's dextrose agar and one of Sabouraud's with penicillin and streptomycin added, with inoculations made in this order. Most slants were streaked about 2 to 4 hours after the swab was taken. To limit the hazard of air contamination all windows were closed, the entire length of the workbench was swabbed with 5 per cent cresol and work was done over paper toweling soaked with 5 per cent cresol. Procedures used for identification of growth included slide mounts, slide cultures, and subculturing to Czapek's medium, potato dextrose agar, corn meal dextrose agar, and corn meal plates.

Problem cultures, both bacteriological and mycological, were submitted to the Laboratory Service Division, Communicable Disease Center, U. S. Public Health Service, Atlanta, Georgia, for study. Dr. N. F. Conant's laboratory, Duke University, also accepted problem mycological cultures for identification.

The technic employed for the sensitivity tests has been described elsewhere.[3]

TABLE I.

A COMPARISON OF BACTERIA ISOLATED FROM 1,377 NORMAL AND 646 INFECTED EXTERNAL AUDITORY CANALS.

| ORGANISM | NORMAL EARS NUMBER | NORMAL EARS PER CENT | INFECTED EARS NUMBER | INFECTED EARS PER CENT |
|---|---|---|---|---|
| *Pseudomonas* group | 14 | 1.0 | 423 | 65.5 |
| *Proteus* group | 5 | 0.4 | 53 | 7.9 |
| *Aerobacter aerogenes* | 64 | 4.6 | 174 | 26.9 |
| *Alcaligenes* group | 22 | 1.6 | 43 | 6.7 |
| *Escherichia* group | 8 | 0.6 | 39 | 6.0 |
| Paracolon group | 5 | 0.4 | 6 | 0.9 |
| Alpha-type *Streptococcus* | 24 | 1.7 | 22 | 3.4 |
| Beta-type *Streptococcus* | 0 | 0 | 25 | 3.9 |
| Gamma-type *Streptococcus* | 15 | 1.1 | 49 | 7.6 |
| Beta-hemolytic *Staphylococcus albus* | 801 | 58.8 | 174 | 12.5 |
| Non-hemolytic *Staphylococcus albus* | 467 | 32.5 | 81 | 12.5 |
| Beta-hemolytic *Staphylococcus aureus* | 128 | 9.3 | 64 | 9.9 |
| Non-hemolytic *Staphylococcus aureus* | 90 | 6.5 | 40 | 6.1 |
| Beta-hemolytic *Staphylococcus citreus* | 19 | 1.4 | 3 | 0.4 |
| Non-hemolytic *Staphylococcus citreus* | 15 | 1.1 | 1 | 0.1 |
| Gram-negative chromogenic rods | 34 | 2.5 | 4 | 0.6 |
| Sporeformers only | 182 | 13.2 | 0 | 0 |
| Totals | 1,893 | 137.4 | 1,201 | 185.9 |

FLORA OF NORMAL AND DISEASED EARS

The bacteriological data accumulated in the Jacksonville and Tampa laboratories were strikingly similar with one exception. The Jacksonville laboratory did not identify diphtheroids; the Tampa laboratory did. These were found more commonly in the normal ear. Since there was this discrepancy in observation, and since the diphtheroids appear to be common inhabitants of the normal ear and without etiological significance, the data concerning these are omitted.

The bacteriological observations are given in Table I. Since from one to four organisms were isolated from positive cultures, the num-

TABLE II.

A COMPARISON OF FUNGI ISOLATED FROM 1,366* NORMAL AND 646 INFECTED EXTERNAL AUDITORY CANALS.

| SPECIES | NORMAL EARS NUMBER | NORMAL EARS PER CENT | INFECTED EARS NUMBER | INFECTED EARS PER CENT |
|---|---|---|---|---|
| *Acrostalagmus* | 2 | 0.1 | 0 | .0 |
| *Acrotheca* | 0 | .0 | 1 | 0.2 |
| *Alternaria* | 57 | 4.1 | 1 | 0.2 |
| *Aspergillus* | 53 | 3.9 | 82 | 12.8 |
| *Candida* (not *albicans*) | 40 | 2.9 | 111 | 17.3 |
| *Cephalosporium* | 3 | 0.2 | 0 | .0 |
| *Coniothyrium* | 10 | 0.7 | 0 | .0 |
| *Cryptococcus* | 1 | 0.1 | 14 | 2.2 |
| *Curvularia* | 84 | 6.1 | 4 | 0.6 |
| *Epidermophyton (floccosum)* | 0 | .0 | 2 | 0.3 |
| *Fusarium* | 9 | 0.7 | 0 | .0 |
| *Fusidium* | 1 | 0.1 | 0 | .0 |
| *Helminthosporium* | 5 | 0.4 | 0 | .0 |
| *Heterosporium* | 5 | 0.4 | 0 | .0 |
| *Hormodendron* | 49 | 2.8 | 3 | 0.5 |
| *Montospora* | 2 | 0.1 | 0 | .0 |
| *Mucor* | 2 | 0.1 | 3 | 0.5 |
| *Nigrospora* | 7 | 0.5 | 0 | .0 |
| *Oospora* | 1 | 0.1 | 0 | .0 |
| *Paecilomyces* | 2 | 0.1 | 1 | 0.2 |
| *Penicillium* | 24 | 1.8 | 7 | 1.1 |
| *Pestalozzia* | 3 | 0.2 | 0 | .0 |
| *Phoma* | 3 | 0.2 | 0 | .0 |
| *Pullularia* | 3 | 0.2 | 1 | 0.2 |
| *Rhizopus* | 3 | 0.2 | 5 | 0.8 |
| *Rhodotorula* | 6 | 0.4 | 1 | 0.2 |
| *Scopulariopsis* | 0 | .0 | 5 | 0.8 |
| *Spegazzinia* | 3 | 0.2 | 1 | 0.2 |
| *Stemphylium* | 6 | 0.4 | 0 | .0 |
| Sterile Dematiaceae | 68 | 5.0 | 14 | 2.2 |
| Sterile Moniliaceae | 48 | 3.6 | 10 | 1.6 |
| *Streptomyces* | 8 | 0.6 | 0 | .0 |
| *Syncephalastrum* | 1 | 0.1 | 2 | 0.3 |
| *Trichoderma* | 13 | 1.0 | 7 | 1.1 |
| *Xenosporella* | 1 | 0.1 | 0 | .0 |
| Overgrown by yeast | 1 | 0.1 | 1 | 0.2 |
| Totals | 524 | 38.3 | 276 | 42.7 |

*Eleven additional cultures were taken which were unsatisfactoy for mycological examination.

ber of organisms is greater than the total cultures taken. The percentage distribution indicates the proportion of cultures yielding that organism alone or in combination with others. In some cases, there was a wide difference in the findings for normal and infected ears. Members of the *Pseudomonas* group were found in only 14 (1 per cent) of the cultures of normal ears, but *Pseudomonas* organisms were isolated from 423 (65.5 per cent) of the infected ears. Moreover, in the former, there were only scattered colonies while, from infected ears, the plates would often show a heavy or almost pure culture of these organisms. All other gram-negative rods were identified more frequently in the infected than in the normal ears. The alpha and gamma streptococci were encountered infrequently in the normal ears, and the beta type was not found in these cultures. In many instances, they were recovered from the infected ears, but even here these organisms were not common invaders. The staphylococci, in contrast, were commonly isolated from the normal ear but they were identified less frequently from the infected ears. Difficulty in isolating these from infected ears was related in part to the heavy growth of the gram-negative organisms. Staphylococci, the chromogenic rods, the sporeformers, and diphtheroids, although found in the sparse growth from the normal ear, were often obscured by an overgrowth of other organisms common in the infected ear.

Up to the past decade otitis externa was called "otomycosis." The earlier literature on this condition, reported that mycotic infection was the cause. Our mycological findings are given in Table II. It is evident that the mycological flora is diverse both in the normal and in the infected ears. Representatives of 35 species were identified, 30 in the normal and 18 in the infected ears. Moreover for one of the genera *Aspergillus,* there were 12 species. There were 1,377 cultures from the normal ear of which 11 were unsatisfactory for mycological examination. Of the remainder, 416 (31 per cent) yielded 524 fungus isolations. There were 646 specimens from infected ears and 245 (38.1 per cent) yielded 276 isolations. Only three species occurred with significantly greater frequency in the infected ears—*Candida* not *albicans, Aspergillus,* and *Crytococcus.* Even the infected ears were not often positive. The per cent of cultures yielding these three species were 17, 13, and 2, respectively. Some species were found more commonly in the normal ear, notably *Curvularia* (6.1 per cent normal and 0.6 per cent infected) and *Alternaria* (4.1 per cent normal and 0.15 per cent infected). The remaining species were encountered only occasionally, with about equal frequency in the normal and the infected ear.

TABLE III.

GRAM-NEGATIVE BACTERIA AND STREPTOCOCCI OCCURRING IN THE ABSENCE OF AND IN THE PRESENCE OF OTHER GRAM-NEGATIVE BACTERIA AND STREPTOCOCCI IN 646 CULTURES OF INFECTED EARS.

| | | Associated Organism(s) | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|
| | | Pseudomonas | | Proteus | | Other gram-negative bacilli | | Streptococci | |
| Organisms | Without associated organisms | Only | And others | Only | And others | One only | And others | Only | And others |
| *Pseudomonas* | 211 | .... | .... | 14 | 19 | 128 | 39 | 30 | 24 |
| *Proteus* | 13 | 14 | 19 | .... | .... | 5 | 18 | 2 | 3 |
| Other gram-negative bacilli | 44 | 128 | 39 | 5 | 18 | .... | 12 | 18 | 23 |
| Streptococci | 20 | 30 | 24 | 2 | 3 | 18 | 23 | .... | .... |

TABLE IV

FUNGI OCCURRING IN THE ABSENCE OF AND IN THE PRESENCE OF DEMONSTRATED GRAM-NEGATIVE BACTERIA AND STREPTOCOCCI.

| | Associated organisms | | | | |
|---|---|---|---|---|---|
| Species | None | *Pseudomonas* | Other gram-negative bacilli | Streptococci | Total |
| *Candida* | 13 | 80 | 17 | 1 | 111 |
| *Aspergillus* | 18 | 48 | 14 | 2 | 82 |
| *Cryptococcus* | 3 | 6 | 3 | 2 | 82 |

The combinations of bacteria found in the same culture are shown in Table III. It is noted that *Pseudomonas* only was isolated from 211 cultures while of the other six varieties of gram-negative rods and the three types of streptococci only 77 in all occurred as the sole organism with possible etiological significance. There were multiple combinations for some organisms so the number of cultures shown in the table exceeds the total number of isolations. Table IV relates certain bacteriological and mycological findings for the three species of fungi isolated more frequently from the infected ears. With few exceptions these occurred in association with *Pseudomonas* or other gram-negative organisms, and rarely with streptococci. There was no such association in 13 cultures yielding *Candida* species not *albicans*, and in 18 positive for *Aspergillus*.

These data provide a basis for the consideration of etiology. The mycological findings are predominantly of a negative nature. In our series if any fungi have even a minor etiological role they would be the *Candida* species, not *albicans*, and the *Aspergillus* species. The variety of *Aspergillus* most commonly associated with disease processes elsewhere is *A. fumigatus*. However, in this series only one of this more pathogenic variety was identified among the 80 isolated from infected ears which were classified. (In three instances the species was undetermined.) Actually there were only two isolations of known pathogenic fungi, the *Epidermophyton floccosum*, and in both cases there were associated gram-positive bacilli and streptococci. In one instance, rash had been observed earlier on the groin, but the other case presented no comparable spontaneous complaint and the sailor had shipped out before the cultural examinations were completed. Since the fungi isolated were predominantly a wide variety of the saprophytic organisms commonly found on decaying vegetation, in the upper layers of the soil and in house dust, their occasional presence in the normal and diseased ear is understandable. This does not explain the more frequent occurrence of three species in the infected ears. If these have a primary or secondary etiological role, they numerically would still be a minor cause of this disease entity.

There is, by contrast, substantial positive evidence that *Pseudomonas* is the most common and most important etiological agent. Organisms of this group were rarely found in the normal ear but were present, commonly in large numbers, in the exudate of the infected ear. Their pathogenic property is acknowledge. The organisms were isolated from 65.6 per cent of the cultures from infected ears. In some cases other varieties of gram-negative organisms

TABLE V.

EFFICACY OF SEVEN THERAPEUTIC AGENTS TESTED ON BACTERIA FROM NORMAL AND INFECTED EARS.

| Source of cultures and classification of bacteria | Relative sensitivity | Sulfa-diazine | Penicillin | Dihydro-streptomycin | Neomycin | Aureo-mcyin | Chloro-mycetin | Terramycin |
|---|---|---|---|---|---|---|---|---|
| *Normal ears* | VS | 46 | 0 | 31 | 22 | 28 | 31 | 77 |
| Gram-negative rods | MS | 4 | 4 | 60 | 91 | 73 | 46 | 52 |
| | R | 102 | 148 | 61 | 39 | 51 | 75 | 23 |
| Gram-positive cocci | VS | 152 | 1037 | 646 | 839 | 1244 | 473 | 1451 |
| | MS | 15 | 503 | 661 | 653 | 141 | 498 | 50 |
| | R | 1395 | 372 | 265 | 70 | 157 | 594 | 61 |
| *Infected ears* | VS | 385 | 0 | 79 | 30 | 57 | 75 | 392 |
| Gram-negative rods | MS | 30 | 0 | 183 | 295 | 200 | 127 | 236 |
| | R | 327 | 742 | 480 | 393 | 485 | 540 | 114 |
| Gram-positive cocci | VS | 63 | 132 | 195 | 206 | 364 | 138 | 413 |
| | MS | 7 | 115 | 132 | 137 | 50 | 175 | 10 |
| | R | 308 | 231 | 151 | 119 | 64 | 182 | 65 |

VS, very sensitive MS, moderately sensitive R, resistant

were readily isolated and apparently were present in large numbers in the exudate. Members of the *Proteus* group were isoalted from 0.4 per cent of the cultures of normal ears but from 7.9 per cent of the infected ears, a twentyfold increase. The other gram-negative organisms also were found more frequently in the diseased ear. The evidence concerning streptococci is of a similar nature, but they were isolated from a relatively small percentage of the total cases and, in all but 20 instances, were found in association with gram-negatve organisms. There was no evidence to suggest that other bacteria had any etiological importance.

Thus our cultural evidence indicates that under conditions existing at this location, at this season, and in this group of individuals, otitis externa is predominantly a problem of infection by gram-negative bacilli, chiefly the *Pseudomonas* group. The streptococci probably have a primary or secondary etiological role in some cases. The mycological flora appears to have limited, if any, etiological significance. Certainly on the basis of present evidence the sound therapeutic approach would be to use measures effective primarily in inhibiting the growth of *Pseudomonas* but active also on other gram-negative bacilli and streptococci.

### IN VITRO SENSITIVITY TESTS

Sensivitiy tests were performed by the disc method as described elsewhere.[3] Each organism was classified as very sensitive, moderately sensitive, or resistant. In all, observations were obtained on 3,071 organisms, including those isolated from the normal ear. A summary of findings is given in Table V. It is apparent that the relative sensitivity of these organisms is essentially the same whether obtained from the normal or the infected ear.

Conclusions relative to the anticipated value of therapeutic preparations are indicated more reliably by findings on the probable primary or secondary pathogens isolated from the infected ears. These data are summarized in Table VI. Two preparations, sulfadiazine and terramycin, have the highest promise for *Pseudomonas* infections. Only a few strains of *Proteus* were sensitive to the preparations tested, although streptomycin and neomycin might be beneficial in about one-third of the cases. Considering the other gram-negative organisms, terramycin gave the best in vitro results while all the others tested, except penicillin, apparently would have therapeutic value in some of the cases. For the streptococci, terramycin, followed closely by aureomycin had the most favorable action. A high proportion of these cocci were resistant to sulfadiazine as well as to penicillin.

TABLE VI.

EFFICACY OF SEVEN THERAPEUTIC AGENTS TESTED ON POSSIBLE PATHOGENS ISOLATED FROM INFECTED EARS.

| Organisms | Relative sensitivity | Sulfa-diazine | Penicillin | Dihydro-streptomycin | Neomycin | Aureo-mcyin | Chloro-mycetin | Terramycin |
|---|---|---|---|---|---|---|---|---|
| *Pseudomonas* | VS | 330 | 0 | 16 | 5 | 10 | 16 | 222 |
| | MS | 20 | 0 | 48 | 96 | 72 | 18 | 163 |
| | R | 73 | 423 | 359 | 307 | 341 | 389 | 38 |
| *Proteus* | VS | 3 | 0 | 2 | 1 | 0 | 0 | 2 |
| | MS | 4 | 0 | 15 | 18 | 0 | 6 | 1 |
| | R | 46 | 53 | 36 | 34 | 53 | 47 | 51 |
| Other gram-negative rods | VS | 51 | 0 | 61 | 24 | 47 | 59 | 166 |
| | MS | 6 | 0 | 118 | 178 | 127 | 101 | 71 |
| | R | 205 | 262 | 83 | 51 | 88 | 102 | 25 |
| Streptococci | VS | 15 | 21 | 8 | 3 | 68 | 23 | 81 |
| | MS | 3 | 14 | 4 | 4 | 18 | 21 | 5 |
| | R | 78 | 63 | 84 | 85 | 10 | 52 | 10 |

VS, very sensitive MS, moderately sensitive R, resistant

This summary of total findings indicates only the proportion of cases which might be expected to respond favorably to particular therapeutic agents. The needs of each individual case must be the concern of the physician. Some cases yielded *Pseudomonas* strains which were resistant in vitro to both terramycin and sulfadiazine. Others were apparently due to less common etiological agents which varied markedly in their vitro sensitivities. For a portion of the cases the therapy of choice would be indicated only by cultures and sensitivity studies on organisms isolated from the individual case.

The initial treatment must be based on a knowledge of the most probable etiology, including its most probable sensitivity to therapeutic agents. The drug of choice in the series of cases studied would be the one to which the highest proportion of the *Pseudomonas* are sensitive, but which is active also against other gram-negative bacilli and streptococci. On the basis of these criteria terramycin is the drug of choice. However, a high proportion of the *Pseudomonas* were very sensitive to sulfadiazine also. Thus a combined therapy with terramycin and sulfadiazine should afford maximum relief in the greater number of cases.

The smaller number of atypical infections provide the therapeutic problems. Initial cultures on all cases for sensivitity tests would be helpful and are indicated. If the case did not respond promptly to the initial therapy, evidence would be at hand to indicate the treatment of choice in that particular case.

## COMMENT

In a literature with many contradictions, there is unanimity of opinion that otitis externa has a characteristic seasonal and geographic distribution. It is a disease of the summer months and is particularly prevalent in hot, humid climates. In this country it occurs more commonly in Southern states; elsewhere the American authors have emphasized its high prevalence in the South Pacific area. Two predisposing factors are mentioned repeatedly, swimming and trauma. The demonstration of *Pseudomonas* species in water of some bathing beaches has been suggested as an explanation of the infections, but other than this, cultural studies do not aid in explaining the seasonal and geographic distribution, nor do they throw light on the question of predisposing causes.

The earlier reported investigations of etiology have directed attention to fungi. Prior to the studies by, and on, military personnel in the South Pacific the prevailing opinion was that otitis externa was predominantly a fungus infection. Recent studies including

our own fail to provide evidence in support of this view. It is genrally concluded at present that the importance of fungi, in causing this condition, has been greatly overemphasized. The gram-negative flora has been viewed with increasing interest, and the prominent role of the *Pseudomonas* species has been stressed in many of the more recent reports. The findings presented here serve to support and extend these earlier studies. Again the reports in the literature and our own observations lead to the opinion that under certain undefined conditions in the external ear, organisms not usually considered as pathogens— particularly gram-negative bacilli other than *Pseudomonas*—multiply freely and appear to have etiological importance.

The findings as well as the technics of this study provide a sound approach to therapy. The organisms which are abnormally prevalent in the diseased external ear may be assumed to be of primary or secondary importance etiologically. Therapeutic agents which inhibit the growth of these will favor a rapid return to normal. On the basis of total findings the organisms usually responsible and agents ordinarily effective against these have been determined. Lacking laboratory findings in the individual case, these therapeutic agents will be the drugs of choice. Our data clearly point to the need for a clinical trial and evaluation of a combined terramycin and sulfadiazine therapy. They also indicate the necessity of laboratory control of treatment. The practical approach which appears indicated is to treat immediately with the agents which are usually effective, and later in unresponsive cases to redirect in accordance with findings by cultural and sensitivity studies on the individual case. If terramycin and sulfadiazine clinically are the drugs of choice, then the best routes of application must be determined. No secure conclusion relative to therapy can be stated prior to the completion of extended therapeutic trials.

## SUMMARY AND CONCLUSIONS

1. Cultures from 1,377 normal and 646 infected external auditory canals were examined bacteriologically and mycologically.

2. *Pseudomonas,* other gram-negative bacilli, and streptococci were identified rarely from the normal but frequently from the infected ears. There was no increase in prevalence of other bacteria in the infected ears.

3. The *Pseudomonas* group of organisms appeared to be the most important etiological agent, but other gram-negative bacilli and streptococci evidently had etiological significance also.

4. Mycological flora was sparse and diverse and appeared to have limited etiological significance.

5. In vitro sensitivity tests performed on all bacteria isolated pointed to terramycin and sulfadiazine as the drugs of choice for otitis externa.

6. Initial cultures and sensitivity studies are indicated to guide therapy in the less typical cases.

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XXV

# THE INFLUENCE OF AIRPLANE NOISE ON AUDITORY THRESHOLDS

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There has been a great deal of controversy regarding the degree and permanence of hearing impairment which occurred as a result of exposure to airplane noise. Grow and Armstrong,[1] Guild,[2] Bunch[3] Dickson[4] and Campbell,[5] among others, called attention to the deleterious effect of repeated prolonged exposure to aircraft noise upon the organ of Corti. Bunch[3] demanded assurance that accidents in commercial airplanes were not due to the inability of "old-time" pilots to hear the beam or the radio under difficult atmospheric conditions and emphasized the lack of careful studies in this field.

Bauer[6] stated unequivocally that pilots without ear protection would become deaf while Walter[7] and Wodak[8] were of the opinion that there was invariably a loss of hearing function in older pilots. Other writers emphasized the localization of aviation auditory defects in the region of 4096 cps. Many additional studies reported auditory changes observed in pilots after the completion of long periods of service in the Air Forces or with commercial airlines.

On the other hand, Scott,[23] Poppen,[24] Ranken[25] and others believed that the greatest percentage of hearing loss resulting from exposure to aircraft noise was of a temporary nature and only a small percentage of ears would show permanent damage after exposure to such acoustic trauma. Greene[30] and MacFarland, Graybill and Liljencrantz,[31] Graebner[20] and Heyden[17] denied any considerable degree of deafness in commercial pilots.

Merick,[28] Campbell and Hargreaves[10] and Eldredge and Parrack[32] reported isolated cases where threshold determinations were made before and after periods of exposure to actual aircraft noise.

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The data in this report was obtained while the author was a member of the staff of the AAF School of Aviation Medicine, Randolph Field, Texas.

As a consequence of the small number of controlled studies, it has been difficult to evaluate the changes caused by aircraft noise alone and to differentiate these variations from those produced by previous otitic infections, gunfire or medications.[33]

In cooperation with the Army Air Forces Training Command, it was decided to select a group of enlisted trainees at pre-flight training, obtain their auditory thresholds and then re-examine the hearing of this same group following the completion of each phase of training, i.e., after primary, basic and advanced flight training.[34-36]

One hundred students who had successfully passed their Army physical examinations and had records of 20/20 hearing for whispered voice were examined at pre-flight school. The ages of the trainees ranged from 18-27 years with a mean age of 21 years. Ninety-eight precent of the men had had ten hours or less in the air. At the time of the examination there had been no exposure to airplane noise during the preceding 30 days.

All of the auditory thresholds were made with a Maico D-5 audiometer which had been calibrated and then checked for purity of tone and accuracy of sine wave. Variation in the apparatus was ruled out by calibration with a General Radio 759 B Sound Level Meter both before and after testing procedures at each school. Auditory threshold determinations were performed mainly at night when all laboratory and office activity had ceased and the base noise level was reduced to 25-30 decibels. The audiometric "zero line" was accepted as the threshold of hearing and the nine frequencies from 256 to 11,584 cps were tested

In the initial examination a preliminary audiometric test was given by an assistant in order to familiarize the subject with the testing procedure, and all subsequent threshold determinations in each case were made by the author. Because training schedules could not be interrupted, it was not possible to obtain the desired periods of freedom from aircraft noise.

### OBSERVATIONS DURING AND AFTER PRIMARY TRAINING

All flying in the primary course was done in PT-19A aircraft (175 horsepower engines). This type of aircraft is not equipped with a canopy, but students were required to wear helmets containing puffs which fitted over the auricles and partially protected the ears against ambient noise. These puffs were perforated by right angle tubes which were attached to a Gosport communication apparatus. Measurements of noise levels in the rear cockpit of this aircraft, at an altitude of 5000 ft., showed approximately 110 decibels

TABLE 1.
NUMBER AND PERCENTAGE OF EARS SHOWING DEGREE OF THRESHOLD ELEVATION AFTER EXPOSURE TO AIRCRAFT NOISE DURING PRIMARY TRAINING.

| MAXIMUM THRESHOLD ELEVATION AT ANY FREQUENCY | RIGHT EARS | | LEFT EARS | | TOTAL EARS | |
|---|---|---|---|---|---|---|
| | NUMBER | PER CENT | NUMBER | PER CENT | NUMBER | PER CENT |
| 0 or 5 db elevation | 39 | 52.7 | 33 | 44.6 | 72 | 48.7 |
| 10 db | 18 | 24.3 | 30 | 40.5 | 48 | 32.4 |
| 15 db | 11 | 14.9 | 8 | 10.8 | 19 | 12.8 |
| | | | | | | 18.9 |
| 20 db or more | 6 | 8.1 | 3 | 4.1 | 9 | 6.1 |
| TOTAL | 74 | 100.0 | 74 | 100.0 | 148 | 100.0 |

of noise in a protected position, and 120 decibels when the microphone was placed in the slipstream. Stalling, stunting, gliding and a change in altitude of 1000 ft. caused little variation in the intensity of the noise.

During this training period a mild epidemic of upper respiratory infections occurred with 51 subjects reporting mild to moderate attacks of rhinopharyngitis. Several mild cases of aero-otitis media were reported with prompt relief of symptoms following treatment. No cases of otitis media or purulent sinusitis were reported by the Flight Surgeon or the subjects and at the time of examination all acute respiratory infections had subsided.

The follow-up determinations were made after nine weeks of primary training during which all subjects flew a minimum of 50 hours and a maximum of 70 hours. At the completion of this period 25 men had been "washed out" for various reasons and had been transferred out of flight training. One cadet was on leave. Thus, 74 students (148 ears) of the original group were available for recheck and comparison. A period of four to six hours was allowed to elapse following the exposure to airplane noise, and then a single test was performed by the author.

Figure 1 shows the threshold variations which occurred in the right and left ears in seven of the frequencies tested following the

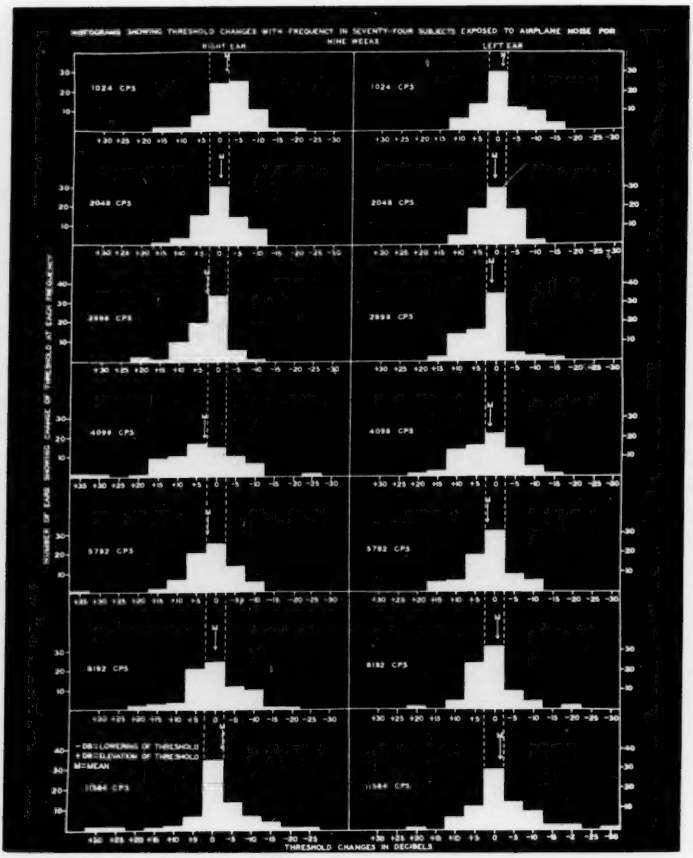
HISTOGRAMS SHOWING THRESHOLD CHANGES WITH FREQUENCY IN SEVENTY-FOUR SUBJECTS EXPOSED TO AIRPLANE NOISE FOR NINE WEEKS
RIGHT EAR
LEFT EAR
1024 CPS
2048 CPS
2896 CPS
4096 CPS
5792 CPS
8192 CPS
11584 CPS
NUMBER OF EARS SHOWING CHANGE OF THRESHOLD AT EACH FREQUENCY
- DISCLOSEMENT OF THRESHOLD
+ DISTELEVATION OF THRESHOLD
M=MEAN
THRESHOLD CHANGES IN DECIBELS

Fig. 1.

TABLE 2.
NUMBER AND PERCENTAGE OF FREQUENCIES SHOWING THRESHOLD ELEVATION AFTER EXPOSURE TO AIRCRAFT NOISE DURING PRIMARY TRAINING.

| | RIGHT EARS | | LEFT EARS | | TOTAL | |
|---|---|---|---|---|---|---|
| | NUMBER | PER CENT | NUMBER | PER CENT | NUMBER | PER CENT |
| Frequencies tested (148 ears x 9 frequencies) | | | | | 1332 | ---- |
| Frequencies showing 5 db elevation | 123 | 9.2 | 118 | 8.9 | 241 | 18.1 |
| Frequencies showing 10 db elevation | 40 | 3.0 | 56 | 4.2 | 96 | 7.2 |
| Frequencies showing 15 db or more elevation | 30 | 2.3 | 13 | .9 | 43 | 3.2 |
| Frequencies showing threshold elevation | 193 | 14.5 | 187 | 14.0 | 380 | 28.5 |

completion of primary training. The two lowest frequencies tested were omitted. There is a real tendency for the more severe elevations in threshold to be associated with the sound frequencies 2896, 4096 and 5792 cps. This is also apparent in the deviation of the mean (M). No such association was evident among the frequencies which showed lowering of thresholds.

Analysis of the thresholds of all the retested ears (Table 1) reveals that 48.7 per cent of the frequencies showed a maximum rise of five decibels; 32.4 per cent showed a maximum rise of ten decibels; 12.8 per cent demonstrated a 15 decibel increase while the remaining 6.1 per cent showed elevations of 20 decibels or more. There is no significant difference in thresholds between the right and left ears.

Examination of Table 2 reveals that 28.5 per cent of all frequencies tested (148 ears x 9 frequencies = 1332 frequencies) show some elevation in threshold on the repeat auditory tests after completion of primary pilot training. Of this number, 18.1 per cent of the frequencies show five decibel elevation; 7.2 per cent show an elevation of 10 decibels; 3.2 per cent show rises of 15 decibels or more.

TABLE 3.
NUMBER AND PERCENTAGE OF EARS SHOWING THRESHOLD ELEVATION AT EACH OF THE FREQUENCIES TESTED AFTER EXPOSURE TO AIRCRAFT NOISE DURING PRIMARY TRAINING.

| FREQUENCIES | MAXIMUM ELEVATION 5 DB | | 10 DB | | 15 DB OR MORE | | EARS SHOWING ELEVATION | |
|---|---|---|---|---|---|---|---|---|
| | NO. | PER CENT | NO. | PER CENT | NO. | PER CENT | NO. | PER CENT |
| 256 | 15 | 10.1 | 2 | 1.4 | .... | ..... | 17 | 11.5 |
| 512 | 14 | 9.5 | 3 | 2.0 | 1 | .7 | 18 | 12.2 |
| 1024 | 21 | 14.1 | 8 | 5.4 | 2 | 1.4 | 31 | 20.9 |
| 2048 | 35 | 23.6 | 8 | 5.4 | 1 | .7 | 44 | 29.7 |
| 2896 | 36 | 24.3 | 24 | 16.2 | 5 | 3.4 | 65 | 43.9 |
| 4096 | 33 | 22.3 | 24 | 16.2 | 15 | 10.1 | 72 | 48.6 |
| 5792 | 38 | 25.7 | 13 | 8.8 | 9 | 6.0 | 60 | 40.5 |
| 8192 | 30 | 20.3 | 6 | 4.1 | 5 | 3.3 | 41 | 27.7 |
| 11584 | 18 | 12.2 | 8 | 5.4 | 5 | 3.3 | 31 | 20.9 |

Table 3 reveals the number and percentage of ears showing rises at any single frequency. It is evident that there is a fairly uniform distribution of the five decibel increases among the high frequencies tested. However, there is a noteworthy difference in distribution when ears showing rises in the threshold of 10 and 15 or more decibels are selected. A significant elevation occurs around 4096 cps and then tapers off above and below this point.

## CONCLUSIONS AND COMMENTS

1. Pure tone auditory threshold tests were obtained on 100 enlisted trainees who had had no recent exposure to airplane noise.

2. Threshold determinations were repeated on 74 subjects (148 ears) who had successfully completed nine weeks of primary training.

3. Because of the training schedule, it was not possible to obtain more than four to six hours of freedom from aircraft noise prior to the hearing retest. Therefore, strict comparison of thresholds obtained before and after exposure to airplane noise during primary training cannot be made.

TABLE 4.
NUMBER AND PERCENTAGE OF EARS SHOWING HEARING LOSS FOLLOWING BASIC PILOT TRAINING

| HEARING LOSS AT ANY FREQUENCY | RIGHT EARS | | LEFT EARS | | TOTAL | |
|---|---|---|---|---|---|---|
| | NUMBER | PER CENT | NUMBER | PER CENT | NUMBER | PER CENT |
| 0 Loss | 11 | 17.2 | 7 | 10.9 | 18 | 14.1 |
| 5 db | 21 | 32.8 | 29 | 45.3 | 50 | 39.1 |
| 10 db | 18 | 28.1 | 17 | 26.6 | 35 | 27.3 |
| 15 db | 7 | 10.9 | 8 | 12.5 | 15 | 11.7 |
| | | | | | | 19.5 |
| 20 db or more | 7 | 10.9 | 3 | 4.7 | 10 | 7.8 |

4. 18.9 per cent of the re-examined ears showed an elevation of threshold of 15 decibels or more at one or more of the nine frequencies tested. This elevation occurred most often at the frequencies 2896, 4096 and 5792 cps.

### OBSERVATIONS AFTER BASIC TRAINING

This study deals with 64 of the original 100 subjects who completed basic pilot training. Each flew 50 to 70 hours under the usual plan of instruction and, at the time of the retest at basic school, each subject had accrued approximately 136 hours of flying time since the original test at pre-flight school. The BT-13A aircraft, a canopied single engine monoplane of 450 horsepower (rated), was the only airplane used during basic training. Noise levels obtained in the front cockpit of this type of plane were found to be approximately 114 db. Indicated altitude was 4000 ft., indicated airspeed, 130 m.p.h., 2000 RPM. Modification of apparatus and calibration was made by Major H. B. Peugnet, MC, AAF School of Aviation Medicine, Randolph Field, Texas.

Because training schedules could not be interrupted, most auditory thresholds were obtained two to eight hours after the completion of flights. This is generally considered an inadequate period of rest following prolonged exposure to aircraft noise. Consequently, all findings in this sample should be interpreted in the light of possible hearing losses resulting from temporary cochlear fatigue.

NUMEROGRAM CORRELATING THE AUDITORY THRESHOLD CHANGES OF 64 STUDENTS FOLLOWING PRIMARY AND BASIC PILOT TRAINING (RIGHT EARS)

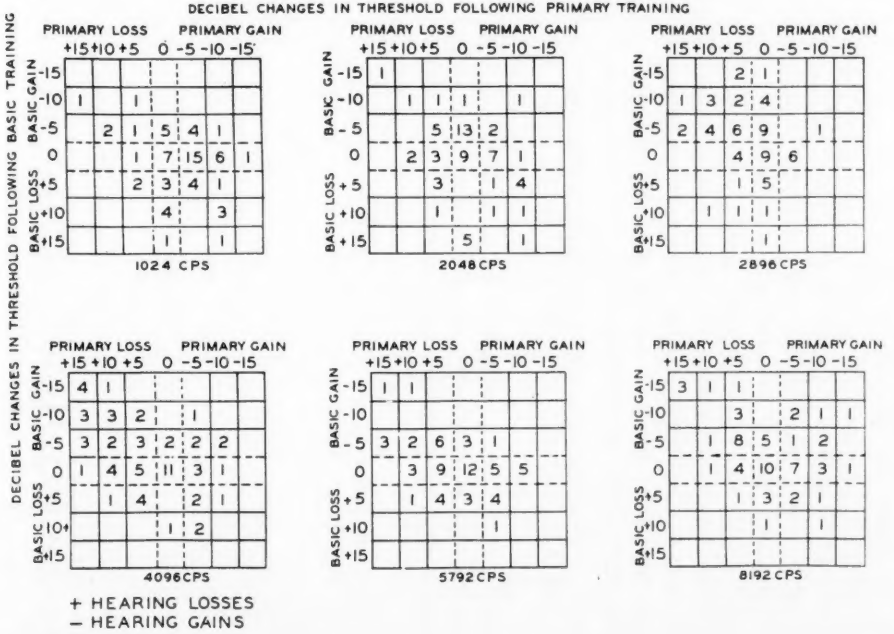


Fig. 2.

TABLE 5.
NUMBER AND PERCENTAGE OF EARS SHOWING HEARING LOSS AT EACH OF THE FREQUENCIES TESTED AFTER BASIC PILOT TRAINING.

| FREQUENCIES | MAXIMUM HEARING LOSS 5 DB | | 10 DB | | 15 DB OR MORE | | EARS SHOWING HEARING LOSS | |
|---|---|---|---|---|---|---|---|---|
| | NO. | PER CENT | NO. | PER CENT | NO. | PER CENT | NO. | PER CENT |
| 256 | 13 | 10.1 | 2 | 1.6 | 2 | 1.6 | 17 | 13.3 |
| 512 | 15 | 11.7 | 7 | 5.5 | 4 | 3.1 | 26 | 20.3 |
| 1024 | 13 | 10.1 | 12 | 9.4 | 3 | 2.4 | 28 | 21.9 |
| 2048 | 12 | 9.4 | 12 | 9.4 | 11 | 8.6 | 35 | 27.4 |
| 2896 | 24 | 18.8 | 7 | 5.5 | 9 | 7.0 | 40 | 31.3 |
| 4096 | 28 | 21.9 | 19 | 14.8 | 4 | 3.1 | 51 | 39.8 |
| 5792 | 34 | 26.6 | 18 | 14.0 | 6 | 4.7 | 58 | 45.3 |
| 8192 | 20 | 15.6 | 8 | 6.3 | 1 | 0.8 | 29 | 22.7 |
| 11584 | 9 | 7.0 | 7 | 5.5 | 7 | 5.5 | 23 | 18.0 |

When the pre-flight and basic hearing thresholds are compared (Table 4) following the completion of basic pilot training, it is seen that 39.1% of the ears examined showed a 5 db elevation of threshold (hearing loss), 27.3% showed 10 db and 19.5% showed more than 10 db at one or more of the frequencies tested.

An analysis of the frequencies at which the losses occurred (Table 5) showed that there was a tendency for the 5 db losses to develop in the region 2896-8192 cps, for the 10 db losses to localize at 4096 and 5792 cps and the 15 db or more group to centralize slightly at 2048 and 2896 cps.

When the losses in the basic sample are compared with those previously reported for the primary sample, a small increase in the percentage of ears (18.9% to 19.5%) showing 15 db or more impairment is observed. The greatest involvement of the frequency 2048 cps in the basic sample is to be noted.

It was considered desirable to correlate for each subject the changes in audiometric threshold obtained after primary pilot training with those demonstrated after basic training. In Figure 2 the

changes in auditory threshold found after primary training are charted on the abscissa and those found after basic training are recorded on the ordinate. Elevation of threshold (hearing losses) are designated positive (+) and lowering of threshold (hearing gains) are labeled negative (—). Each frequency is charted separately. Only the figures for the right ears are given since similar results were obtained in the left ears.

As an example of the interpretation of Figure 2, if at 4096 cps four right ears show a loss of 15 db after primary and a 15 db gain after basic training, then the numeral "4" is placed in the square corresponding to the +15 on the abscisa and −15 on the ordinate. Thus, if hearing losses occur in the primary period and are associated with hearing gains in the basic period there will be a concentration of numerals in the left upper quadrant. Losses developing at primary school and increasing at basic school will be indicated by a large number of figures appearing in the left lower quadrant.

From this data it is seen that there is a tendancy for ears that showed hearing losses in the primary sample to have hearing gains in the basic sample. This tendency is evident in all the higher frequencies and is particularly marked at 2896 and 4096 cps. A small number who showed gains after primary training demonstrated losses after basic, and another small group showed losses in both samples.

These results are borne out in histograms of the 64 subjects (Figure 3) which show the threshold changes occurring at each frequency from the pre-flight to the basic sample. When contrasted with previous observations comparing the pre-flight and primary samples, it is seen that the average losses sustained at primary training were, to a large extent, recovered at the time that retests were performed at basic training.

The apparent differences which appear in the findings when the percentage of ears showing 15 db or more losses is compared with the average deviation for all ears (both positive and negative) result from the clinical custom of considering only deviations below the audiometric zero-line (positive variations) and disregarding deviations above the zero-line (negative variations). When all the positive and negative changes in threshold are averaged, we obtain data which suggest that no statistically significant hearing losses persist following basic training. From the otologic viewpoint, however, a relatively large number of ears in this series show hearing losses at one or more frequencies.

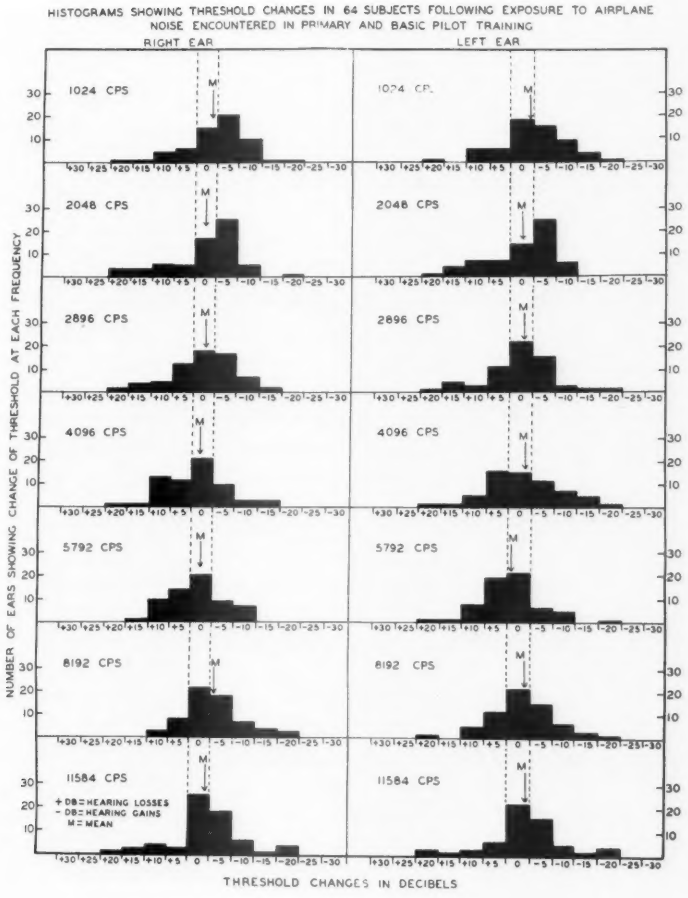
HISTOGRAMS SHOWING THRESHOLD CHANGES IN 64 SUBJECTS FOLLOWING EXPOSURE TO AIRPLANE NOISE ENCOUNTERED IN PRIMARY AND BASIC PILOT TRAINING
RIGHT EAR
LEFT EAR
1024 CPS
2048 CPS
2896 CPS
4096 CPS
5792 CPS
8192 CPS
11584 CPS
+ DB = HEARING LOSSES
- DB = HEARING GAINS
M = MEAN
NUMBER OF EARS SHOWING CHANGE OF THRESHOLD AT EACH FREQUENCY
THRESHOLD CHANGES IN DECIBELS

Fig. 3.

## CONCLUSIONS AND COMMENT

1. Following the completion of approximately 135 hours of flying (primary and basic pilot training), 19.5% of ears showed threshold elevations (hearing losses) of 15 db or more at one or more of the frequencies tested.

2. The greatest percentage of clinically significant losses occurred at the higher frequencies (2048-5792 cps).

3. In many cases, losses incurred during primary training tended to diminish during the basic period, despite continued exposure to aircraft noise.

4. Observations should be interpreted in the light of the fact that most retests were performed two to eight hours after exposure to aircraft noise.

## OBSERVATIONS FOLLOWING ADVANCED TRAINING

This study deals with 54 of the original 100 subjects examined at pre-flight school who had been assigned to advanced pilot training. On the basis of flying aptitude and individual preference for single or multi-engine aircraft, these men had been divided into two groups. Thirty-five were assigned to advance two-engine school and 19 were sent to advanced single-engine school.

Each subject flew approximately 70 hours under the usual conditions of instruction in the advanced training period, and thus accrued approximately 210 hours of total logged flying time. The UC-78 aircraft was used at two-engine school. Overall noise levels obtained at the pilot's left ear were found to be approximately 123 db. Indicated altitude was 3100 ft.; indicated airspeed 130 m.p.h., 1940 RPM. At single-engine school the AT-6 was used. In this aircraft, with the canopy closed, noise levels of 118 db were found, while with the canopy open, overall levels of 128 db were observed in the front cockpit. Indicated altitude was 7000 ft.; indicated airspeed 140 m.p.h., 1875 RPM. Toward the completion of their advanced training, each student in single-engine school flew a small number of hours in the P-40 aircraft.

Thirty-two of the two-engine subjects and eight of the single-engine students (mean age 21 years) had 24 hours or more freedom from aircraft noise prior to audiometric retest. This provided a total of 40 men (80 ears) who had 24 hours or more of freedom from noise prior to retest. This group will hereafter be referred to as the "rested" group. It is not to be assumed that 24 hours of free-

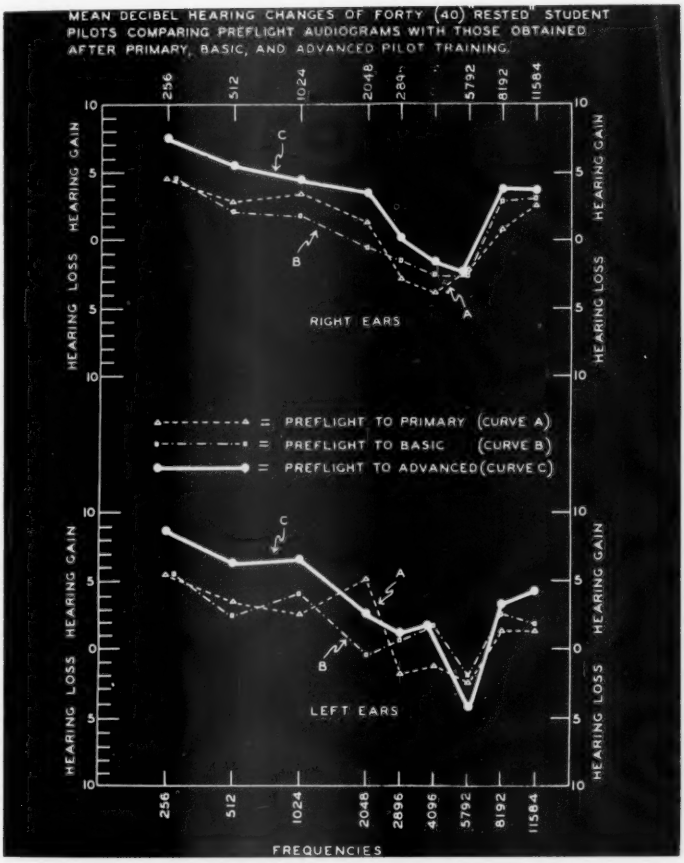
MEAN DECIBEL HEARING CHANGES OF FORTY (40) RESTED STUDENT PILOTS COMPARING PREFLIGHT AUDIOGRAMS WITH THOSE OBTAINED AFTER PRIMARY, BASIC, AND ADVANCED PILOT TRAINING.
HEARING LOSS HEARING GAIN
RIGHT EARS
LEFT EARS
= PREFLIGHT TO PRIMARY (CURVE A)
= PREFLIGHT TO BASIC (CURVE B)
= PREFLIGHT TO ADVANCED (CURVE C)
FREQUENCIES
256
512
1024
2048
2896
4096
5792
8192
11584

Fig. 4.

dom from noise is considered an adequate period of rest for all subjects under all conditions.

Because the training schedule could not be interrupted, 11 of the subjects at single-engine school and three of the men at two-engine school (mean age 21 years) were retested one-half to eight hours following the completion of their last flying mission. This latter group consisting of 14 men (28 ears) will be referred to as the "non-rested" group.

The mean decibel hearing changes for the "rested" group (80 ears) are compared in Figure 4, while Figure 5 shows the same comparison for the "non-rested" group (28 ears). Comparison is made within each group between: A) pre-flight and primary; B) pre-flight and basic; and C) pre-flight and advanced pilot training.

In Figure 4 it is seen that in the case of the "rested" ears, the mean hearing losses which occurred at 2896, 4096 and 5792 cps after primary or basic training (Curves A and B) have been partially recovered after advanced training (Curve C). Only at 4096 and 5792 cps in the right ears and at 5792 in the left ears are residual losses still in evidence following advanced pilot training.

The "non-rested" group of ears offers a striking comparison (Figure 5). Although the curves of the primary and basic groups (Curves A and B) are essentially like those of the larger "rested" group, the mean threshold determinations following advanced training (Curve C) show widespread losses involving the frequencies from 1024 to 5792 cps inclusive. A tendency for mean losses to peak in the region of 4096 and 5792 cps is evident in all groups tested.

In all the curves represented in Figures 4 and 5 there is a tendency for low frequencies to show gains up to nine decibels. This may be explained by the fact that sound isolated rooms were not available and the second audiometric test prior to aircraft noise exposure was accepted as the base threshold for each trainee. Thus, the variation in the mean auditory threshold is in accordance with observations that during audiometric determinations the low frequencies are readily masked by variable ambient noise;[37, 38] also that on repeated testing a more accurate threshold is obtained on the third or fourth audiometric examination.[39]

The large amount of hearing loss found in the "non-rested" advanced group (one-half to eight hours' freedom from noise) is in sharp contrast to the small amount of loss found in audiograms after

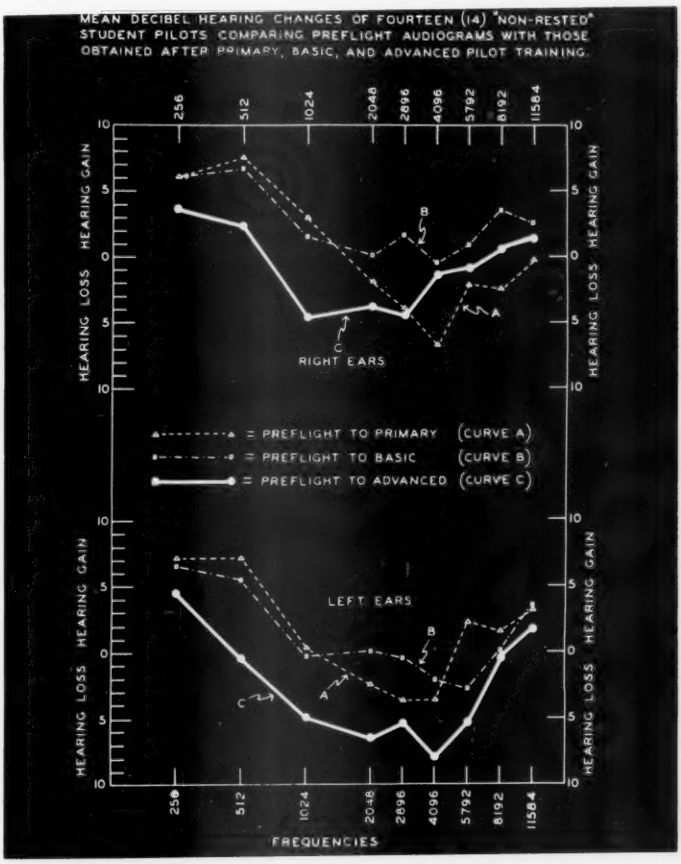
MEAN DECIBEL HEARING CHANGES OF FOURTEEN (14) "NON-RESTED" STUDENT PILOTS COMPARING PREFLIGHT AUDIOGRAMS WITH THOSE OBTAINED AFTER PRIMARY, BASIC, AND ADVANCED PILOT TRAINING.
256
512
1024
2048
2896
4096
5792
8192
11584
HEARING LOSS HEARING GAIN
10
5
0
5
10
RIGHT EARS
A
B
C
= PREFLIGHT TO PRIMARY (CURVE A)
= PREFLIGHT TO BASIC (CURVE B)
= PREFLIGHT TO ADVANCED (CURVE C)
LEFT EARS
FREQUENCIES

Fig. 5.

basic training (two to eight hours) of freedom from noise). A review of the figures reveals that the 14 student pilots comprising the present "non-rested" group had flown an average of two hours seven minutes on the day they were retested after basic training, and had logged an average of two hours 23 minutes on the day of the retest after advanced training.

The very small differences in the duration of exposure and period of freedom from aircraft noise in this group following basic and following advanced training are insufficient to explain satisfactorily the marked difference in the degree of hearing loss. Davis and coworkers,[40] Perlman[41] and others[42, 43, 32] have shown that temporary hearing losses may be produced by stimulation with pure tones or synthetic aircraft noise. It has been demonstrated that the distribution of the hearing loss is directly related to the pitch of the fatiguing tone (always occurring at or above the stimulating tone), while the amount of threshold elevation, in general, varies directly with the duration of exposure, the overall intensity of the pure tone or airplane noise and the susceptibility of the subjects.

It would appear then that a difference in the noise spectrum or the overall intensity of the aircraft noise in the more powerful advanced trainers may be sufficient to account for the large amount of aural fatigue found among the "non-rested" advanced students. After a period of 24 hours or more of freedom from noise, the perception of all of the lower tones and some of the higher tones rapidly returns to normal.

## CONCLUSIONS

1. Fifty-four of the original 100 subjects examined at preflight school successfully completed advanced pilot training. Forty of these subjects were then given audiometric retests following a period of freedom from noise of 24 hours or more ("rested" group). Fourteen subjects had one hour to eight hours from aircraft noise prior to retest ("non-rested" group).

2. Among the "rested" ears, mean hearing losses sustained during primary or basic pilot training were partially or completely recovered following advanced training.

3. In striking contrast, the "non-rested" ears of the advanced group showed mean hearing losses which were widespread, involving the frequencies 1024 to 5792 cps inclusive.

SUMMARY

1. Pure tone auditory thresholds were obtained on 100 subjects prior to pilot training and after each successive phase of their training, namely, primary, basic and advanced.

2. It would appear from the foregoing that a large percentage of men in pilot training will show temporary hearing losses following the usual period of exposure to aircraft noise.

3. These temporary losses tend to involve the frequencies 1024 to 5792 cps.

4. In the more vulnerable 2896 to 5792 cps region, recovery of hearing losses is delayed.

5. It may be concluded that hearing losses incurred during primary, basic or advanced pilot training are, on the whole, temporary losses which are completely recovered following 24 hours of freedom from exposure to aircraft noise.

500 N. Skinker Blvd.

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XXVI

# FURTHER CLINICAL INVESTIGATION ON THE EFFECT OF DRAMAMINE UPON COCHLEAR FUNCTION

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AND

JOSEPH SATALOFF, M.D.

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Gay and Carliner have first shown dramamine (B-dimethylaminoethyl benzohydryl ether 8-chlorotheophyllinate) to be extremely valuable in the prevention and treatment of motion sickness.[1] It appears to be of value occasionally in relieving the vertigo in prolonged attacks of Ménière's symptom complex. Campbell has found dramamine to be effective in modifying the vertigo and spontaneous nystagmus resulting from the reactive serous labyrinthitis which usually immediately complicates the fenestration operation for otosclerosis.[2]

One of us has found that dramamine prevents the nauseant and emetic effect of morphine.[3, 4] Pharmacologically it appears to be an antiacetylcholine agent and it is through this probable action that it prevents the nauseant and emetic effect of morphine, the latter drug being a very potent anticholinesterase agent in brain tissue.[3] Dramamine appears to exert this antiacetylcholine effect wherever and whenever acetylcholine activity is present above a normal physiological level. In this clinical experiment[3] the effect of dramamine upon the vestibular responses in normal subjects and its effect upon hearing was noted. Eleven medical students served as subjects.

The hearing was checked before and after the oral administration of 200 mg of dramamine in a sound conditioned room by Miss Marian Knorr. The same audiometer was employed throughout these studies. In two of the 11 subjects there was a drop in hearing which increased at and above 1024 double vibrations per second. Repetition of the audiograms several days later revealed a return of the hearing to normal in each subject. Three weeks later audiograms

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From the sections of Neuro-otology and Audiology of the Department of Otolaryngology, University of Pennsylvania, School of Medicine.

were repeated without and with 200 mg of dramamine in one of these two subjects. (The second subject did not make himself available for a repetition of this experiment). The audiograms were found to be normal in each instance. We were at a complete loss to explain the hearing impairment in these two subjects after the first trial with dramamine.

It was, therefore, deemed advisable to continue this experiment using a greater number of subjects in order to arrive at a more definite conclusion on this point.

In our present experiment 46 normal medical students served as subjects. The hearing of each subject was checked with the audiometer in a sound conditioned room. One hundred mg of dramamine was then taken orally. One hour later a second audiogram was made. This was immediately followed by the oral administration of a second 100 mg of dramamine. One hour later, i.e. two hours after the first 100 mg of dramamine, a third audiogram was made. In no instance was there any change in the hearing.

## COMMENT

If dramamine caused the temporary hearing impairment in two of 11, or 18%, of our subjects in our original investigation, we should have been able to demonstrate such an effect upon repetition of the experiment in these two subjects. Audiograms before and after repeat oral administration of dramamine in one of these two subjects were normal.

Furthermore, in a group of 46 subjects we should anticipate an effect upon hearing by dramamine approximately eight times. Actually, we were unable to demonstrate a cochlear functional effect by dramamine in any of our 46 subjects.

## CONCLUSION

It appears that dramamine has no effect upon hearing.

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XXVII

# PRELIMINARY REPORT ON AN APPLICATION OF THE MOTOR FUNCTION OF THE SUPERIOR LARYNGEAL NERVE

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In view of the importance of surgical correction for bilateral adduction of the vocal cords in man, the innervation of the vocal cords was studied in the dog.

*Method.* Dogs were anesthetized by intraperitoneal nembutal, and the field exposed through a midline cervical incision. The superior laryngeal nerves were followed with the help of the fenestration loupe to their point of penetration of the cricothyroid muscles and there cut. The recurrent laryngeal nerves were sectioned just below the cricoid cartilage. The faradic current was used as a stimulus. The vocal cords were observed with the laryngoscope.

*Results.* Section of both recurrent laryngeal nerves in the dog is followed by bilateral adduction of the vocal cords. Simultaneous bilateral faradic stimulation of that branch of the superior laryngeal nerve which supplies the cricothyroid muscles is followed by blanching, tensing, elongation, and further adduction of the vocal cords.

Section of both recurrent laryngeal nerves and the right superior laryngeal nerve as it enters the cricothyroid muscle is followed by adduction on the left; but on the right side, on which the motor division of the superior laryngeal nerve has been divided, the vocal cord is in the cadaveric position.[1] Such relaxation of the vocal cord permits a good glottic chink and a fair bark in the dog.

If studies now in progress on patients indicate that the motor division of the superior laryngeal nerve has a function similar to that described for the dog, then these findings suggest the use of this procedure for the correction of bilateral adduction of the vocal cords,

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instead of any of the various operations for fixation of the arytenoid cartilage.

THE JOHNS HOPKINS HOSPITAL.

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# XXVIII

# STUDIES ON THE OTIC LABYRINTH

## I. ON THE ORIGIN AND INSERTION OF THE TECTORIAL MEMBRANE

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The attachments of the tectorial membrane have been the subject of study and speculation for a great many years. The origin of the tectorial membrane on the limbus extending from the margin of the lamina osseae to the vestibular lip of the limbus seems to be well established. An insertion at the outer margin has been suspected by many and, though attempts have been made to demonstrate it, the matter still remains in doubt. The tectorial membrane is usually pictured as having its attachment at the limbus and then floating free above the hair cells a varying distance. Some maintain that it is in contact with the hair cells, and others feel that it floats above them with the hairs making contact during vibration of the basilar membrane by being lifted upward until they strike the under surface. DeVries[2] states that the hair cells are in contact but that this contact can be readily broken by touching with a needle. He found no other attachment. He describes a marginal net thrown back over the upper surface of the tectorial membrane, which was evident only with phase contrast microscopy.

Mygind[7] in studying histologic preparations found the tectorial membrane in extensive contact with the organ of Corti but he considers this an abnormal phenomenon caused by compression. Kolmer[6] speaks of a "randfasernetz," sometimes found at the outer margin of the tectorial membrane, which he thinks is probably attached to the reticular membrane of the "papilla basilaris" (organ of Corti). After carefully dissecting the tectorial membranes from pig embryos, Hardesty[3] concluded that the outer margin was free, that the outer zone overlay the entire organ of Corti and that, in this zone, there is a net on the under surface which can be peeled off. He found the

Work done as visiting research professor at Columbia University, Department of Otolaryngology, and supported in part by grants from the Research Council of the American Otological Society and by the Hayden-Coakley Memorial Fund.

membrane slightly elastic longitudinally and very flexible transversely.[3] V. Held,[4] who made extensive studies of the cochlea, including the tectorial membrane, describes the "randfasernetz" and states that in the embryo this might be attached but after birth the last fibers let go. Bethes[1] mentions the "randfasernetz" and states that it has been seen by Retzius, Held, Satoh (who considered it an artifact) and others.

Not all research workers on the ear have considered the outer margin of the tectorial membrane as free and unattached. Shambaugh[9] and Prentis[8] accepted the view that the hair cells were attached. The former maintained that the hairs penetrated the surface of the tectorial and that in addition, Hensen's stripe is attached to a row of cells inside the inner hair cells—the so-called border cells. Both Kishi[5] and Wittmaack,[10] working on laboratory animals, demonstrated connections between the organ of Corti and the tectorial membrane. For some reason these studies do not seem to have been generally accepted, at least as far as man is concerned.

The anatomy of the tectorial membrane with its attachments assumes a position of paramount importance if one is to understand the physiology and pathology of hearing. It seems to be fairly well accepted that the stimuli from the mechanical vibrations of sound waves are applied to the sense cells at the tectorial membrane, but, how this is done, still remains unexplained.

## STUDY ON THE ORIGIN AND INSERTION OF THE TECTORIAL MEMBRANE

*Material and Methods:* The material used in this study consisted of 121 temporal bones, 90 of which were human and 31 of which were removed from freshly butchered pig embryos. The latter were considered unsatisfactory for the purposes of this study because of differences in anatomy and, when 21 had been used, the other 10 were discarded. Of the specimens from man, 74 were removed by the author at autopsy as soon after death as possible. Some came from infants (prematures, stillborn or dead neonatally from other causes) but most were from adults. The other 16 from man were removed from preserved heads of infants which were already in the laboratory. With the exception of ease of dissection in the specimens from infants, there seems to be no essential difference between the otic labyrinths of adults and infants. Most of the specimens removed at autopsy were dissected and studied fresh within 24 hours after the autopsy, or, if not disected immediately, they were kept under refrigeration. If it was not possible to make the study

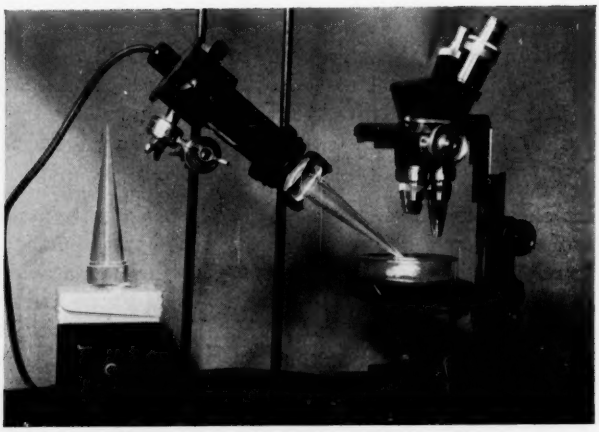

Fig. 1.—One of the Lucite cones used for illumination in this study illustrating manner of use. It was turned and polished in a machinist's shop.

within 24 hours after autopsy, the specimens were placed in ten per cent formalin.

The aim has been to make fresh dissections, avoiding histologic procedures which might alter delicate structures. Only a few simple stains were applied directly to the fresh tissues.

Most dissections and examinations were made under a Greenough type of dissection microscope. To overcome the obstacle of insufficient light in the cochlea, a lucite cone, turned out and polished on a machinist's lathe, was used* (Figs. 1 and 2). The light is very intense within a few millimeters of the tip and can be used either as transmitted or reflected light, either in air or submerged under Ringer's solution. Such a cone can be used to carry light through an enlarged internal acoustic meatus, through windows cut into various turns of the bony cochlea or directly through the thin endosteal bone.

*Findings:* The tectorial membrane has a firm origin on the upper surface of the limbus, which is in the form of a sheet or membrane that fuses with or is attached to the upper ends of the cells composing the limbus (Fig. 3). This limbal zone (or zone of origin) of the tectorial membrane has three subzones which differ somewhat. The innermost of these lies at the inner margin of the limbus

*This will be described in detail in a subsequent article.

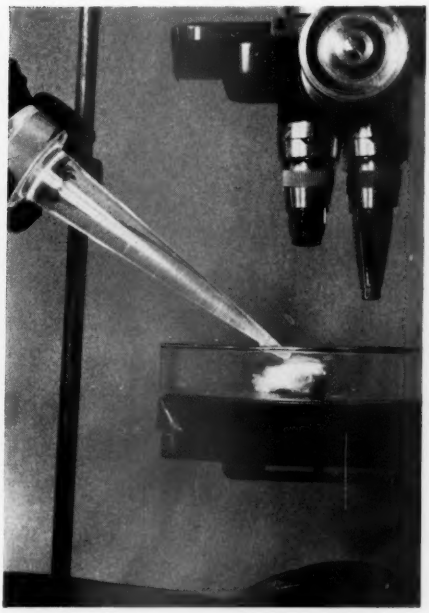

Fig. 2.—Close-up of Fig. 1 showing lighting of submerged specimen.

next to the lamina osseae and is more or less bar-like (anchor bar) with stubby anchors at close intervals along the inner edge (Fig. 4). The next subzone is the thinnest and weakest band and lies next outside the "anchor bar." It comprises, with the bar, perhaps one third of the width of the limbal zone. If a tear occurs, when the tectorial membrane is separated from this limbal origin, it will be along this thin inner third. The third subzone is thicker and more firm and comprises about two-thirds of the limbal zone. It is in this region that the oblique fibrils in the upper portion of the tectorial membrane arise and pass in a tangential direction of 30 to 45 degrees with the long axis, outward and toward the apex, to be inserted in the outer margin—what is now called the "free" outer margin. Fibrils may and probably do originate as far in as the anchor bar but the author has not been able to visualize them with certainty.

This limbal portion is attached to the underlying cells by a cement or glue-like material which stains purple with methylene blue. As the limbal portion is separated from the underlying cells, it tends to carry cell ends with it or to form interstices where the cell ends tear out thus making this limbal zone into a more or less

Fig. 3.—View across detached tectorial membrane. O zone of origin on the upper surface of the limbus. In situ it appears to be an intact membrane. When detached, it often appears lacy. The interstices probably represent thin spots overlying cell ends which tear during removal. It may be that some of the material in the membrane clings to the underlying cells and is torn out, just as cell ends sometimes cling to the membrane and are torn off the cells. IM-remnants of the membrane of insertion clinging to the outer margin. H. S.—Hensen's stripe. In fresh specimens this appears to be made up of the same homogeneous substance found in the outer margin and in the net-like remnants of the membrane of insertion. The dark bodies contained within it are cell remnants. Focal plane at the inferior surface.

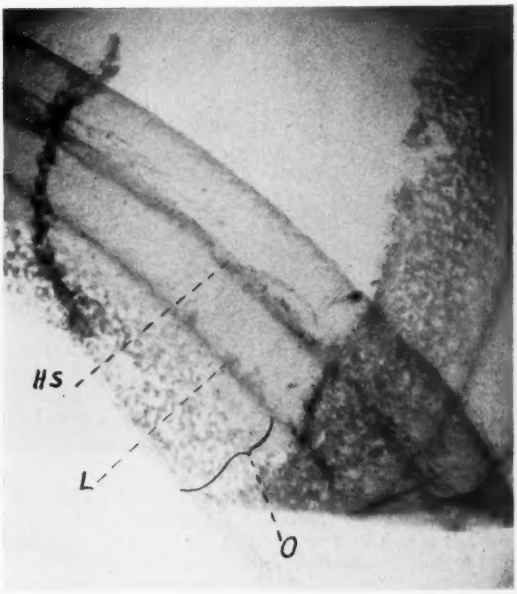


Fig. 4.—Folded tectorial membrane showing the lacy zone of origin with the anchor bar partly detached. The region just outside the bar is the weakest band where this zone generally tears. Longitudinal folds can be seen faintly just outside Hensen's stripe. O—Zone of origin. L—Line of attachment at lip of limbus. H. S.—Hensen's Stripe.

lacy structure (Fig. 3). One sometimes gets the impression that the material in the membrane contracts or condenses into cords, which has the effect of changing the intact membrane into a lacy one.

In addition to the insertion on the upper aspect of the limbus, some fixed specimens show what seems to be a similar but smaller insertion on the inner wall of the inner sulcus which joins with that just described at the lip (vestibular) of the limbus. This second origin is not regularly found and could be either a variation or an artefact. Where it occurs the wedge-like lip of the limbus splits the origin of the tectorial membrane into a "Y" which can be seen readily in fixed preparations. There is an accumulation of substance—called "cement substance" for want of a better term—on the margin of the limbal lip. It seems to be quite flexible.

*Insertion:* The tectorial membrane normally has an insertion as well as an origin, the insertion being on the organ of Corti. Be-

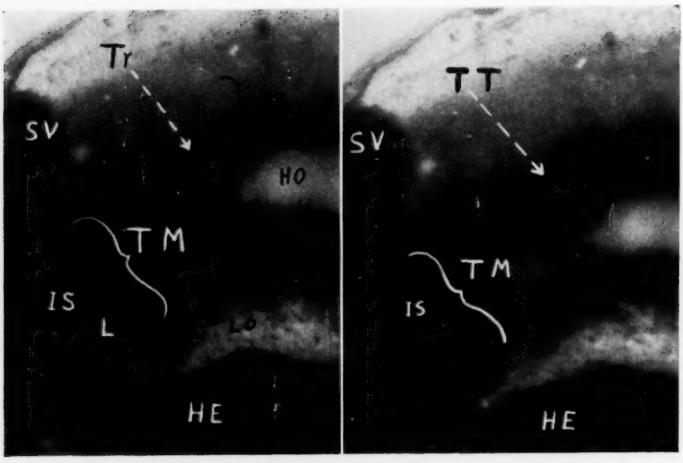


Fig. 5.—View looking into apical turn of the cochlea. The intact tectorial membrane has been engaged by a tiny hook—HO, held in a micromanipulator and is making traction upon the organ of Corti into which it is inserted. It can be seen that the latter yields and follows the line of traction. By manipulating the hook, both the organ of Corti and the basilar membrane were rocked back and forth within the limits set by the checking action of the spiral ligament. HE—helicotrema. L—limbus. LO—lamina ossea. IS—internal sulcus. SV—stria vascularis. Tr—point of traction. OR—organ of Corti. TM—tectorial membrane.

Fig. 6.—Same specimen a few minutes later, after a tear in the tectorial insertion had occurred from the manipulations (T.T.).

cause it is difficult to expose this insertion without rupturing it, it is exceedingly difficult to demonstrate. I have succeeded in doing so, to a varying degree, in at least twelve specimens. In no specimen was the insertion completely intact, but some attachments were visualized in each of these twelve. In a few the insertion (or insertions) seemed to be nearly complete and intact. The normal intact insertion is firm enough so that it will lift and rotate the entire organ of Corti and basilar membrane as far as the supporting attachments (they might be called "check ligaments") of the latter permit (Figs. 5 and 6).

The insertion is not simple but complex and difficult to make out. It seems to consist of at least four elements or planes, which fuse together more or less completely, and might be designated as follows:

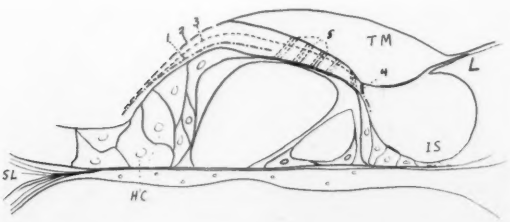


Fig. 7.—Diagrammatic representation of the insertion of the tectorial membrane as the author reconstructs it separated out to show the different planes. 1) Layer which sometimes during dissection tears at the base of Hensen's cells. It extends inward at least as far as the hair cells and perhaps all the way to the internal sulcus. 2) Marginal membrane or net extending to the base of Hensen's cells. The "Randfasernetz" of Held is a torn remnant of this. 3) Highly distensible layer which probably has a roller bearing action and may be confined in vivo to the region of the sense cells. 4) Attachment at Hensen's stripe to the border cells in the internal sulcus. 5) Hair cell attachment.

(1) A continuous sheet or membrane which extends inward from the slope of Hensen's cells across the top of the organ of Corti. How far inward it extends I do not know.

(2) A sheet or net originating in the outer margin of the tectorial membrane and passing outward over the bulge of the organ of Corti as far as the base of Hensen's cells.

(3) Long tongue-like processes of cement substance attached to the under surface of the tectorial membrane which pass outward transversely across the organ of Corti to its outer slope where they seem to end.

(4) An attachment between Hensen's stripe and the border cells (Figs. 5 and 6) just inside the inner hair cells. The first three may be and probably are fused together from the outside line of outer hair cells to the base of Hensen's cells.

The first of these four elements (the membrane which extends across the organ of Corti) stains poorly with methylene blue or Mallory's connective tissue stain. Under certain circumstances, it lets go at the outer base of Hensen's cells and pulls upward and inward forming ballooning folds. In specimen No. 117 it peeled inward as far as the tunnel cap where it floated (the specimen was submerged in Ringer's solution) nearly upright in festooning folds, like an inverted drape.

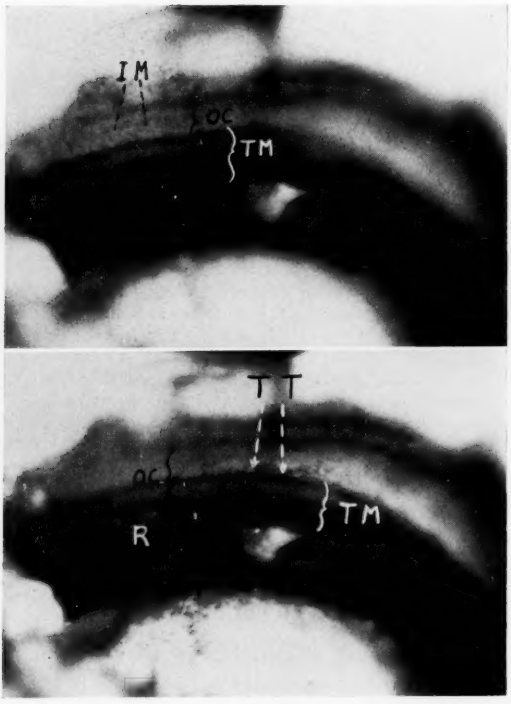


Fig. 8.—Tectorial membrane (TM) attached upon the organ of Corti (OC) by membrane of insertion which appears like a faint, fine net spreading outward from the tectorial margin—IM.

Fig. 9.—Point further along toward the apex showing the outer margin of the tectorial membrane tearing free and carrying some of the insertion with it in the form of a net—TT. R—remnant of Reissner's membrane.

The second element (the sheet or net originating in the outer margin of the tectorial membrane) is composed of a homogeneous material, which stains fairly well with Mallory's connective tissue stain (aniline blue) but is so exceedingly thin as to be practically invisible in many spots. When stained in situ it appears like a faint blue net extending outward from the tectorial margin, over and covering the bulge of the organ of Corti, down to the base of Hensen's cells. (In some specimens, it even seemed to cross the outer sulcus and extend to the prominentia spiralis.) It can be visualized quite well under a magnification of 50 or 100 diameters with a

Fig. 10.—View across organ of Corti. The hair cells and reticulum can be seen in the lower half. A narrow line of the outer margin of tectorial membrane (TM) can be seen pulling away. The dark masses are remnants of the insertion membrane. They have collected thickly along the base of Hensen's cells, which is at the top of the picture.

Greenough dissection microscope (Fig. 8). When the membrane over Corti's organ (just described as the first element of insertion) is detached along the base of Hensen's cells and floats partly free, it carries the net from the margin with it. The author has succeeded a few times in engaging this net or sheet in a microhook and tearing it off Hensen's cells. As soon as it tears free, it contracts into a strand in the manner of a woman's hair net subjected to tension.

A microhook fixed in a micro manipulator was used a number of times for the purpose of engaging the upper surface of the tectorial membrane and manipulating it. When the insertion was intact, as for instance in 102 a, the entire organ of Corti and basilar membrane could be lifted, rotated inward and rocked back and forth within the limits determined by the checking action of the spiral ligament.

Many attempts were made to dissect the organ of Corti together with the tectorial membrane and its attachments out of the cochlea, and to place them on a slide for examination under high magnification. Such a dissection of the cochlear duct with the tec-

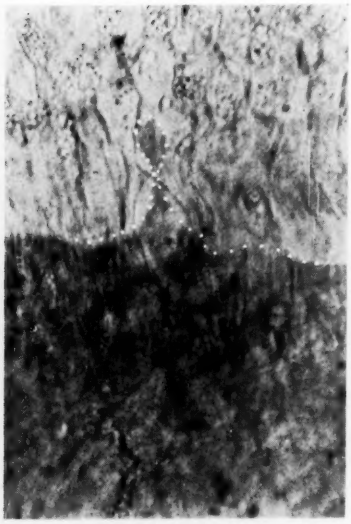

Fig. 11.—View looking down upon the top of the organ of Corti. In the upper half, part of the reticulum and the outer hair cells can be seen. The lower half is seen through the tectorial membrane, the margin of which crosses about the middle. In the center, the margin is drawn into a point where it still remains attached to a small blob of insertion membrane (outlined in white dots). In the fresh stained specimen, which was stained blue, these features showed distinctly.

torial insertion on the organ of Corti intact is fraught with the greatest difficulty. However, this was accomplished more or less successfully a few times (specimen 92, for example). In such preparations, seen under oil immersion lens, this marginal sheet of insertion stained faintly blue and overlay the upper surfaces of the underlying cells. It appeared deepest at the cell margins, whereas over the centers of cells it was so thin as to be practically invisible. This characteristic may give it the appearance of a net when viewed under low power in situ. When mounted on a slide, it tends to contract or condense forming strands or amorphous blue islands (perhaps more accurately described as "pools") (Fig. 10). Some of these seem to have fibrillar connections between the underlying cells. In such a mounted preparation, the outer margin of the tectorial membrane proper is much less definite than when this margin is free. When attached, it seems to feather out thinly to be lost in the sheet of insertion and seems to be under tension and about to tear free (Figs. 10 and 11). In all of these studies, the tectorial membrane seemed to be

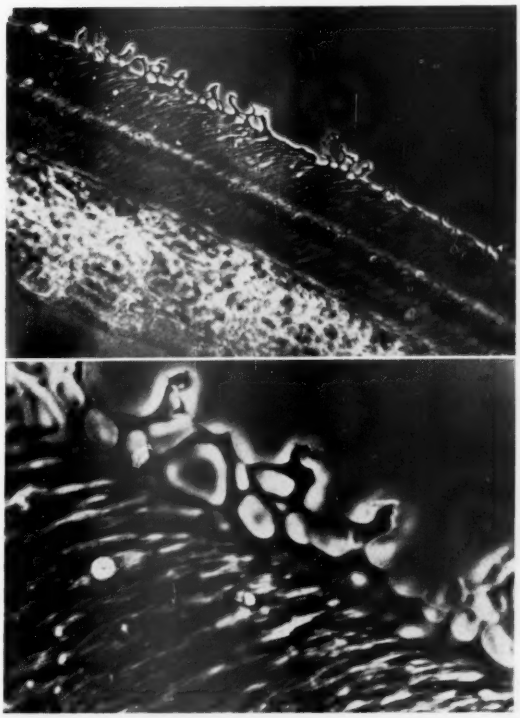

Fig. 12.—View of tectorial membrane by phase contrast microscopy. The blurred zone is the zone of origin. The net-like frayed remnants of the membrane of insertion are well seen.

Fig. 13.—High power view of details at margin. It can be seen that the fibrils lose themselves in the homogeneous material which makes up the margin and then continues out to make up the cords of the fringe. This insertion membrane or net, as it appears here, is probably similar in nature to the zone of origin only much more delicate. When seen in situ it appears like a vanishingly thin membrane. When lifted off the underlying cells, like the zone of origin it cannot maintain itself as a membrane, but becomes like a net. Either the thin material from the interstices condenses into the cords or remains on the cells. One gets the impression that it is all made up of glue-like cement substance.

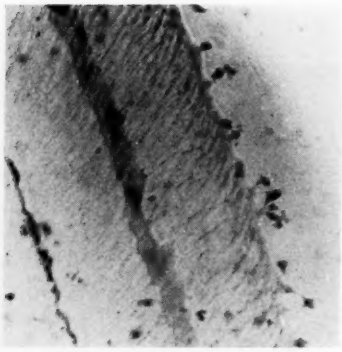

Fig. 14.—A marginal fringe which condensed into olive shaped knobs or balls. This is the only specimen in which this was found. Hensen's stripe shows points of traction on both sides.

under tension unless detached. (A possible explanation for this will be given in a subsequent article.)

The author has succeeded a few times in detaching the tectorial membrane from its origin on the limbus and preserving the insertion on Corti's organ (specimens 105 and 106). When the tectorial membrane is then grasped and torn off the organ of Corti with traction directed up and out, this sheet of insertion is seen to tear along the top of the rounded ridge of Corti, often leaving a frayed edge attached (Fig. 12) to the tectorial margin. Under other circumstances, it tears just as often in the cement substance of the margin leaving the latter perfectly smooth. When a portion of the sheet of insertion remains attached as a frayed edge, it appears like an irregular net. The homogeneous matrix, or cement substance, in the margin passes directly out into and forms the cords of this net (Fig. 13). Whether or not this net is a continuous sheet in situ is not clear. It may be that it is and that the cords of the net represent contractions of material which occur when it is torn loose, in somewhat the same manner in which the limbal origin is filled with lace-like interstices when it is torn loose. In manipulation, however, this net seems to be fairly firm and unchanging. This was true in most of the many tectorial membranes which were studied. One (No. 106) proved to be an exception; in this one, the cords in the frayed marginal net condensed and contracted into rounded or pear-shaped knobs such as soft latex might be expected to form when released from tension (Fig. 14).

Fig. 15.—Marginal fringe from the torn membrane of insertion is faintly visible. Something very similar is seen along the outside of Hensen's stripe. This could be torn remnants of the insertion just inside the inner hair cells described by Shambaugh. Such frayed strands have been seen on the under side of the tectorial membrane a number of times when this surface has been thrown into profile by folds.

The under surface of the tectorial membrane from the internal sulcus to the outer row of outer hair cells is in direct contact with and attached to the cells of this portion of the organ of Corti. Long tongue-like processes extend from the under surface, outward across Corti's organ, where they are lost over Hensen's cells. Some have been followed practically to the base. These presumably constitute the third element of insertion and form the strands which connect the under side of the tectorial membrane with the membrane over Hensen's cells. In one specimen (No. 117) the author drew back and kinked the outer margin of the tectorial membrane until a short loop lay out of contact with Corti's organ. A microhook was then passed under the tectorial membrane where it engaged some strands which, when placed under tension, simultaneously lifted the loose loop of the tectorial membrane and the outer torn border of the membrane overlying Hensen's cells. One end of these engaged strands seemed to be attached to the under surface of the tectorial membrane near Hensen's stripe, whereas the other pulled the detached membrane from a point which had lain (in situ) far out.

This distensible third element may be in the nature of a semifluid or cement substance, the function of which is to permit of free movement between the under surface of the tectorial membrane and the reticulum—a roller-bearing, so to speak.

The fourth element, the attachment to the border cells, is very difficult to visualize. It is the mate to the attachment of the outer border to the cells of Hensen. Shambaugh[9] found it intact in at least one fixed specimen. The writer believes that he has actually seen it once—in specimen 105. Further evidence consists in the following. Numerous cell fragments have been found in the stripe which do not seem to be from hair cells. Attempts to relate the stripe in situ to the inner hair cells have failed. It always lies just inside the hair cells. The mass of border cells is similar to Hensen's cells and occupies a corresponding position on the inside of the fibrous arch of the organ of Corti. Hensen's cell mass is attached to the border through a stripe of homogeneous substance in the border. The substance in the stripe seems to be the same.

In addition to these four elements in the insertion of the tectorial membrane, the hair cells are also connected to the under surface, i.e. either the hair cells themselves or the reticulum in which they are supported. In specimen No. 105, the limbal origin was first separated and then the tectorial membrane was peeled or torn off the organ of Corti by lifting it up and out. The hair cells, each in turn, were seen to lift and stretch as tension was made and then to tear loose and snap back into position. Some left remnants in the tectorial membrane. In this specimen, the strongest line of attachment seemed to be along the line of internal hair cells and was undoubtedly the insertion into the border cells.

When traction is exerted on the tectorial membrane until its insertions are separated, that separation generally follows a certain sequence. The net or sheet extending from the margin to the base of Hensen's cells is rather inflexible and gives way first. It tears either right in the margin close to the insertion of the fibrils, leaving a smooth edge, or along the top of the ridge of Corti's organ, leaving a frayed net attached to the tectorial margin (Fig. 9). The remainder, lying on the outer convex slope of Hensen's cells, tends to gather into blue-staining, transverse strands lying across the outer half of the organ of Corti, or into patches or islands, or to collect at the base of Hensen's cells (Fig. 10). Sometimes strands remain under tension, deeply grooving the organ of Corti. Such blue-staining strands are attached both at the base of Hensen's cells and

near the tunnel cap. This is one indication that all of these parts are more or less fused together.

Next to give way is the attachment of the outer hair cells. When the margin is loose, the later tolerates very little traction. It will lift the organ of Corti but tends to tear readily. This attachment too is not very flexible, whereas, on the other hand, the fibers, or tongues of cement substance, which attach the under side of the tectorial membrane to Hensen's cells seemed, at least in one specimen (No. 117), to be very distensible. In this specimen they were pulled completely outside the bony cochlea, yet continued to lift both the tectorial membrane and the membrane overlying Corti's organ which had been torn loose at the base of Hensen's cells.

More infrequently, when the insertion is torn loose, the membrane just mentioned, overlying the whole of Corti's organ, gives way along the base of Hensen's cells and, floating partly free, rotates inward. Sometimes Hensen's cells seem to burst and the contents balloon out.

Although the insertions of the tectorial membrane seem relatively inflexible, the cells of the organ of Corti, especially Hensen's cells, seem quite mobile. The organ of Corti, as a whole, is a rather firm and closely knit structure, which is not readily torn to pieces. Attempts to tear it apart frequently result in separating it from the basilar membrane as a unit.

## SUMMARY

The tectorial membrane originates on the limbus in an attachment, which is familiar to all who have examined fixed specimens. In fresh specimens, the layer of origin on the upper surface is a membrane, which at times looks lace-like and is attached to the limbal cells by a cement substance. At the inner margin of the limbus, there is a comparatively heavy bar, which seems to be firmly attached, by stubby projections occurring at short intervals, to the lamina ossea or the inner margin of the limbus. The upper fibrils in the thick main portion of the tectorial membrane extend from this limbal origin to the outer margin.

The central thick portion of the tectorial membrane is divided into two more or less equal halves, the inner half spanning the inner sulcus from the lip of the limbus to the line of border cells next inside the inner cells and the outer half extending from these border cells to and including the outer line of outer hair cells. This latter portion of the tectorial membrane is firmly inserted into the

organ of Corti by a rather complex set of insertions containing several planes or elements. It is in intimate and extensive contact with the underlying cells to which it is attached. Long attachments extend from the under surface to the cells of Hensen. There is a membrane (net or sheet) extending from the margin across the organ of Corti to the outer sulcus. Further, there is a heavier and deeper membrane which covers a large part. There is strong evidence also supporting Shambaugh's contention that there is an insertion to the line of border cells just inside the inner hair cells. The tectorial membrane together with Hensen's cells and these border cells, forms and constitutes a flexible arch which fits closely over the relatively inflexible arch that carries the sense cells.

It is doubtful that any of the elements of insertion connect directly with the reticulum or the arch supporting the sense cells.

When intact, the insertion is sufficiently strong so that it can be made to lift and rock the organ of Corti and basilar membrane. When the attachment of the outer margin has ruptured, the remaining attachments can still be made to rock the organ of Corti gently.

The writer is indebted to Dr. Vladimir Epanchin for his patient photographic work, done under most difficult conditions.

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# XXIX

# STUDIES ON THE OTIC LABYRINTH

## II. A THEORY ON THE STIMULATION OF THE ORGAN OF CORTI BY SOUND VIBRATIONS

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The past 300 years have witnessed the development of many theories of hearing, with each proponent of a new concept feeling that he, at last, was on the right track leading to the solution of the problem. Though the writer fully appreciates this and approaches the subject with the feeling of humility which this engenders, since finding that the tectoria is definitely inserted upon the organ of Corti (described in a paper presently in the press), he wishes to submit another version of that part of the hearing problem having to do with the localized stimulation of the organ of Corti by means of widespread vibrations.

Among the many scholarly studies dealing with the vibrations of the basilar membrane are those of Peterson and Bogart,[3] Ranke,[4] Zwislocki,[8] Wegel,[6] and von Békésy.[1] The vibration patterns seem to have been well worked out, and, as far as a non-mathematical mind can assess the results, the investigators agree fairly well. They concur in the opinion that a large part of the basilar membrane vibrates with each tone. Ranke[4] believes that v. Békésy's experiments show beyond a doubt that the pattern of vibration has a flat top but Ranke admits that the ability of the ear to discriminate pitch remains unexplained. Almost without exception, the research workers in this field seem to have considered the up and down movements of the basilar membrane as being the stimulating movement initiating nerve impulses in the sense cells. Wegel[6] speaks of a shearing force derived from the normal motion of the organ of Corti (normal to the basilar membrane) and a synchronized longitudinal movement of the fluid in the scala media. Ter Kuile[5] describes a sliding motion between the tectorial membrane and the hair cells, produced

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by the movements of the inner and outer rods of the tunnel as the basilar membrane rises and falls. Wever,[7] in his book, refers briefly to this idea, but dismisses it in favor of vertical pressure.

These studies of the up and down vibratory movements in the basilar membrane (and organ of Corti) have been so carefully made that it behooves one to accept as fact the patterns reported. If the up and down movements are as widespread as described, then it seems highly unlikely that these are the movements carrying the stimulus to the organ of Corti, and the author feels that this theoretical point might profitably be abandoned. Moreover, since the search for a focal point of movement, which might be stimulating, has thus far proved fruitless, might one not look instead for a focal point in an otherwise moving field which is held relatively still? In pursuing this new line of thought, one must not fail to acknowledge the debt of gratitude which is owed to these careful workers for their very important contribution in the description of the shape of the waves and their positions.

Many facts have been learned about the exceedingly complex mechanism involved in the hearing organ, but, many of the research studies in hearing leave the impression that the investigator has disregarded some obvious and pertinent facts and has freely made assumptions which are not valid anatomically. The solution of the hearing problem seems to lie in the acceptance of the known facts and in their correlation with one another.

A number of facts, receiving little or no attention, seem, to the writer, to be pertinent:

(1) The organ of Corti contains two arches differing widely in mobility and structure; these are the closely knit arches, carrying the sense cells and their supports, and an outer flexible one which fits closely over the first.

(2) The tectorial membrane is supplied with fibrils, extending from the limbal zone upward toward the apex, at a tangent of 30° to 40° with the axis, to be inserted in the outer margin.

(3) As the basilar membrane rises and falls, the inner half swings on an arc pivoted at the tympanic lip of the lamina ossea, or at a point between the lamina and the floor of the inner sulcus.

(4) The organ of Corti, riding upon the basilar membrane, describes the same arc.

(5) The tectorial membrane is pivoted at the lip (vestibular) of the limbus at a point which lies at a higher level than the pivot of the basilar membrane.

(6) The difference in the level between these two points is greatest in the basal turn and smallest in the apical turn and the difference between the base and the apex though not great, seems to be graduated.

(7) The outer margin of the lamina ossea recedes progressively from a plane drawn through the lip of the limbus and vertical to the basilar membrane. It finally ends entirely at the hamulus and its supportive functions are taken over, at least in part, by the taut, fibrous margin of the helicotrema.

(8) The tectorial membrane is more flexible transversely than longitudinally. When point pressure is made upon its upper surface, an oval-shaped dimple results, with its long axis lying parallel with the fibrils.

(9) The outer margin of the basilar membrane is fixed by check ligaments permitting very limited movement.

(10) The tectorial membrane is inserted into a sheet or membrane overlying Hensen's cells, and also into the border cells in the internal sulcus.

(11) The tectorial membrane fits closely down upon the hairs of the hair cells.

(12) When radial traction is made upon a tectorial membrane which is normally inserted upon the organ of Corti, the greatest response in the latter is not in the radius where traction is made, but at a point closer to the apex.

(13) The inner half of the tectorial membrane proper spans a fluid-filled moat—the inner spiral sulcus.

(14) The border cells, just inside the internal hair cells, are very similar to Hensen's cells and form a similar smaller sulcus.

Correlating these facts provides us with a basis for a new departure in the study of the stimulus resulting from sound vibrations in the cochlea. With the tectorial membrane firmly attached to the organ of Corti and riding pick-a-back upon it, it is difficult to believe that the vertical up and down movement is stimulating. From the standpoint of physics, it is difficult to imagine how there could be a vertical pressure (normal to the under surface of the tectorial membrane) upon the ends of the hair cells. The same lifting force transmitted to the hairs of the hair cells from the basilar membrane through the supporting structures, would also be transmitted to the under surface of the tectorial membrane by any liquid which might

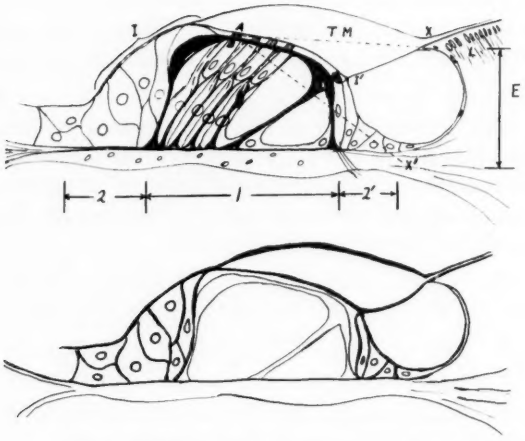


Fig. 1.—(upper) Diagrammatic representation of a cross section of the organ of Corti modified after Held. The firm fibrous arch which carries the sense cells is marked by heavy lines (1) and the flexible arch in faint lines (2,2′). I—zone of insertion of the outer margin of the tectoria (T.M.) I′—insertion at Hensen's stripe into the border cells. X—lip of limbus and pivot point of the tectoria. X′—pivot point of the inner half of the basilar membrane and the organ of Corti. E—elevation between the two pivot points. (lower) Diagrammatic representation of the flexible arch (heavy lines) entirely covering the firm, fibrous arch.

lie between the latter and the reticulum. Both the hairs and the tectorial surface would lift simultaneously and equally.

On the other hand, with the organ of Corti and the tectorial membrane both rising and falling in curved arcs in response to the same force, but each being pivoted at a different level, a shearing force would inevitably occur between them. The result would be a shearing motion unless one or the other were sufficiently flexible to yield and neutralize such motion (Fig. 2) or unless the attachment between them were so firm as to maintain immobility, in which event there would have to be a yielding at some other point.

Let us consider points on the organ of Corti and the lower surface of the tectorial membrane which would be in contact when the basilar membrane is at rest (Fig. 1a, A&B). In up and down motion each would describe its own arc (D&D$^1$ & C&C$^1$) (Fig. 2). The two arcs would intersect at the point of rest and would deviate from one another either above or below this point by a distance dependent upon the length of the arcs (amplitude) and their radii. If two bodies which are in contact are moved on such arcs, the up

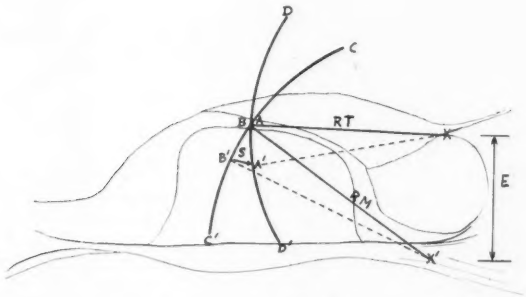


Fig. 2.—Diagram of the movements of the organ of Corti and the tectorial membrane which result in a shearing motion. E—elevation between the two pivot points and X and X.[1] DD[1] arch in which point A on the tectorial membrane swings. RT—radius of that arc. CC[1] arc in which B on the organ of Corti swings. RM—radius of that arc. When point A is depressed to A,[1] then B travels to B[1] causing a shearing force S in the direction of the arrow.

and down force would be translated into a shearing force between them. This shearing force would be greater than the up and down force in inverse proportion to the relation between the length of the radii and the distance between the pivot points. If the radii were equal and long, compared to the distance between the pivot points, there would be a mechanical advantage in favor of the up and down force (small force and large amplitude resulting in a small amplitude and larger force). The closer together the planes of the two pivot points, the greater will be the mechanical advantage and vice versa. If one radius were longer than the other, this would result in a reduced mechanical advantage and a greater deviation between the two arcs. Both amplitude and force would be reduced if the pivot on the shorter radius began to rise and fall somewhat with the longer radius—in other words, to ride upon it.

If these principles are applied to the tectorial membrane and the organ of Corti, it is evident that the distance between the pivots is smaller than the radii upon which the organ of Corti and the tectoria swing. Therefore, the amplitude of the vibratory movement of the basilar membrane is reduced to a shearing motion with smaller amplitude and greater intensity. This change is in the same direction as that occurring between the tensor tympani and the oval window; the amplitude is reduced and the force increased.

The difference in the levels between the two points of pivot (i.e. the height of the lip of the limbus above the basilar membrane)

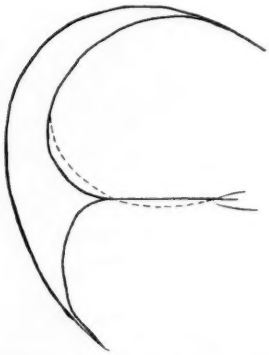

Fig. 3.—Diagram illustrating the checking action of the spinal ligament modified after Meyer. When the basilar membrane has moved downward to the point where it makes, together with the upper limb of the spiral ligament, an "anchor chain" curve, then further movement is resisted.

decreases progressively from base to apex, tending to increase the mechanical advantage, further intensifying the shearing force, and would be in the opposite direction from the increased amplitude and decreased force of the basilar membrane from base to apex, as now understood. However, the outer margin of the lamina ossea recedes further and further from the inner sulcus, thus making the radius of the vibrating basilar membrane longer as compared with that of the tectorial membrane. This would be in the direction of greater amplitude and less force in the shearing movement. Concurrently both radii increase progressively in length from base to apex, lending intensity in the direction of shearing motion again. As the lamina ossea recedes, the exact location of the pivot point on the basilar membrane becomes uncertain. In one specimen, the author observed the movements in the apical turn and the point of pivot appeared to be in the floor of the internal sulcus—well outside the bone. Beyond the hamulus, it becomes increasingly difficult to postulate, from fixed specimens, where the point of pivot would be. The entire supporting structure, including the margin of the helicotrema, is membranous but there is no reason to believe it all vibrates, in the manner of the basilar membrane. If it did, there would be little shearing force and it may be that the narrowed basilar membranes in this region are designed to obviate this factor. All one can say is that a number of factors act in the production and intensity of the shearing force between apex and base and all of them seem to be graduated, but in such a way as to neutralize one another. It may be that

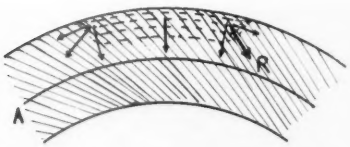


Fig. 4.—Diagram of tectorial membrane with contained fibrils, showing an area pushed down by a sound impulse. The arrows indicate the directions of the shearing forces which would result. The only one parallel to the fibrils is at R. The tectoria is distensible transversely across the fibrils but is not distensible in the direction of the fibrils. If the distensibility across the fibrils is sufficient to neutralize the shearing action, then the only direction where the shearing force would result in movement between the tectoria and hair cells would be that indicated by the arrow R.

the amplitude of the shear is kept rather constant for protective reasons because it seems likely that the movements which the hair cells can tolerate without injury are definitely limited.

Now where does all of this lead us? Of what advantage is a shearing movement over an up and down motion in the solution of our hearing problem? The amplitude of the movement has been decreased but it is just as widespread longitudinally as the vibratory movement of the basilar membrane.

Let us consider an impulse coming through the scala vestibuli from the stapes. The basilar membrane, with its riding structures, begins to move downward. It does not resist greatly at first but as soon as it, together with the upper limb of the spiral ligament (Fig. 3), approaches the "anchor chain" curve, it resists strongly (Meyer[2]). If displacement to this point is insufficient to accommodate the fluid displaced by the inward movement of the stapes, then additional areas of basilar membrane will move downward into the "anchor chain" position until the displacement of fluid has been equalized. Let this hypothetical downward displacement involve one-third of the length of the basilar membrane and remain stationary. In this picture, we find the organ of Corti and the attached tectorial membrane in four different positions (Fig. 4):

(1) the unaffected neutral position

(2) a slope downward facing the apex

(3) a slope downward facing the base and,

(4) a flat depressed region between these two slopes.

The shearing force, through the entire length of the flat depressed region, would result from a pull by the tectorial membrane directed

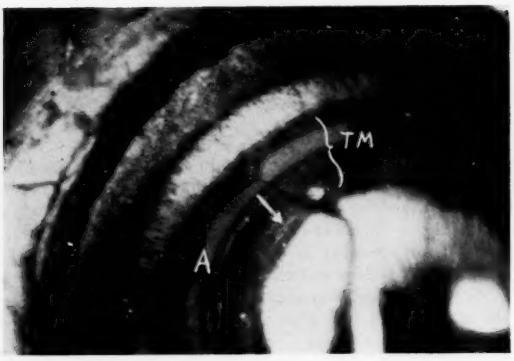


Fig. 5.—View into the apical turn of the cochlea. Action of the tectorial membrane (TM) in response to traction. A microhook is engaged in its upper surface and is making traction as indicated by the arrow (not the axis of the hook). A is toward the apex. It will be noted that the fold which has developed shows more tension toward the apex.

radially toward the modiolus. On the slope facing the apex there would be a force directed radially toward the modiolus. On the same slope, there would be another force directed toward the base because the thickness and mass of the tectoria would resist a bend downward, somewhat after the manner of an axial attachment. If the tectoria were placed directly over the organ of Corti, this force would be exactly axial, in which case, the resultant of these two forces, if equal, would be at 45° between the two, and would be directed inward and toward the base. Since the tectoria is not so placed but instead is on the inside slope of the organ of Corti, the second force would be directed axially and inward and the angle of the resultant force would be reduced to less than 45.° The exact direction could not be determined unless the magnitudes of the two forces and the exact angles of the axial pull were known, but it would probably be in the neighborhood of 25° to 40.° A similar set of forces would act upon the slope facing toward the base and would be directed inward and toward the apex at about the same angle.

If one examines the tectorial membrane, it is seen to contain parallel fibrils passing outward and toward the apex at a tangent. The uppermost fibrils originate upon the limbus, where they seem firmly attached. They insert in the margin of the tectoria about at the line of the outermost hair cells. When the tectoria is normally inserted in the organ of Corti, these upper fibrils are attached at

both ends and one gets the impression that they are under some tension. Presumably the other contained fibrils originate and insert in a somewhat similar manner. In attempting to measure the angle of the fibrils with the axis, I obtained a measurement of about 30.° (It varies with the condition and position of the tectoria.)

By pressing upon the top of the tectorial membrane an oval dimple is produced, the axis of which parallels the fibrils, demonstrating that the fibrils resist the pressure more than the remainder of the substance of the tectoria. Radially directed traction upon the tectoria in situ results in a pull toward the apex rather than radially (Fig. 5).

A number of workers have reported that the tectorial membrane is more flexible transversely than axially. If this transverse flexibility were sufficient (for example, in the zone spanning the inner sulcus) to neutralize the normal range of shearing movements produced by sound vibrations, then shearing force would be ineffective in producing stimuli to the hair cells. According to v. Békésy, as quoted by Perlman, the up and down amplitude of the basilar membrane is in the magnitude of a single micron for ordinary sounds. The shearing movement would be less. It is quite possible that this amount of movement could be neutralized by the flexibility of that portion of the tectoria which spans the inner sulcus. The zone in contact with the sense cells would ride up and down on the organ of Corti with little or no shearing force. If, however, this shearing force were in a direction parallel or nearly parallel with the fibrils, and if these latter are not flexible (as indicated by the oval dimpling and other findings) then they would initiate a shearing movement in their own axis. This would occur, of course, only if they were anchored at their origin and attached to the organ of Corti. They do, in fact, seem to be so anchored at their origin and attached at their insertion. The uppermost fibrils seem to be anchored almost on the bone of the lamina ossea itself. If these fibrils are essentially non-yielding, then the hair cells in the outer line near the point of insertion of the fibrils, would find themselves receiving a jerk in downward movement—a jerk which would come directly from the stationary bone or firm tissue close to it.

On the slope of our hypothetical depressed section of tectorial membrane, which is directed toward the base, the resultant force of shear would be directed toward the apex and inward at an angle of some 30.° However, it would cross the direction of the fibrils at an angle of 60° (if that of the fibrils were 30°), the same angle at which the direction of radial shear crosses them. Again, if the tectoria were sufficiently distensible transversely, there would be no

stimulating shearing movement. Thus, in our hypothetical picture, the only spot on the tectorial membrane which would transmit a stimulating, shearing force to the organ of Corti would be the small area on the slope of the wave which is directed toward the apex.

The position of this sloping effective area in the cochlear coil would depend upon the pitch of the stimuli. It seems well established that it moves toward the apex in low tones and toward the base in high tones.

The insertion of the tectorial membrane is upon the large, globular cells of Hensen, which appear, in fresh specimens, to be highly mobile within a small range. The border cells, just within the hair cells, seem to be of a similar nature. These two masses together with the attached tectorial membrane constitute a flexible arch which, as far as I know, has never been described as an arch. Within this flexible one stands the comparatively inflexible arch that carries the sense cells, and in which they are firmly fixed. The stiff hairs themselves are attached to the under surface of the tectorial membrane and may actually penetrate it. It looks as though the tectorial membrane, although its insertions hold it snugly down upon the hairs, is, nevertheless, because of the mobile character of these insertions, at liberty to rock freely upon the hairs within very limited amplitudes—an excellent arrangement for the transmission of impulses of traction to the hair cells from a distant, relatively stationary point, through the fibrils in the tectorial membrane, which are fixed in origin at that stationary point.

When an impulse passes into the scala vestibulae action results about as follows: 1) A region of the tectorial and basilar membranes together with the organ of Corti is pressed downward. The flexible arch (tectorial membrane, large mass of flexible cells [Hensen's] and small mass of flexible cells [border cells] fitting closely over the supportive arch, rides the latter downward. 2) A resulting shearing force occurs in three directions (a) radially on the flat depressed area (b) inward and toward the apex on the slope facing the base (c) inward and toward the base on the slope facing the apex. 3) The transversely distensible tectorial membrane, especially that part which spans the internal sulcus yields, allowing the flexible arch to ride downward without any shearing movement on the hair cells. This occurs in all areas except in the small area of the slope facing the apex. The force here directed inward and toward the base is applied in the axis of the fibrils which are fixed on the limbus (possibly even on the lamina ossea). Being fixed and nondistensible, they cannot yield.

Instead, 4) they exert a pull on that portion of the tectorial membrane where they are inserted, causing a shearing motion which is transmitted to the underlying hairs of the hair cells. 5) The hairs bend (or are affected by the force in some other manner) in the direction of the pull, (for each set of hairs this pull would always be in the same direction) and a stimulus results. Meanwhile, 6) the insertion of the corresponding part of the tectorial margin is subjected to a pull which is absorbed, 7) by the yielding globular cells in Hensen's group. 8) The downward movement is stopped by the checking action of the upper limb of the spiral ligament before damage is done to the marginal membrane of insertion. 9) The structures rise again in response to the phase of recovery. 10) The tectorial is pulled back into position by the elasticity of Hensen's cells. 11) The negative phase carries the structures above the position of rest until the lower limb of the spiral ligament checks further movement in this direction. One would think from the shape and attachments of the spiral ligament that downward movement is more limited than the upward. The downward movement would make more stress upon the insertion of the tectorial membrane and might do damage if excesive.

Thus, in vibration, the flexible arch rides the non-flexible one like a saddle rides a horse. Meanwhile, the transversely distensible tectorial, accordian-like, takes up the shearing motion, especially in that portion which bridges the internal sulcus. If more space downward is needed here for this changing bridge, the endolymph readily moves axially in both directions.

When, on the other hand, the shearing force is directed along the fibrils, then portions of the tectorial are pulled back in their downward course, causing a shearing movement on the hair cells and a pull on the insertions. This pull is now taken up in the two masses of flexible cells, the larger one in the external sulcus (Hensen's cells) neutralizing the pull in the larger excursion and the small mass of cells, in the internal sulcus neutralizing the pull in the smaller excursion close to the pivot point.

This hypothetical picture is undoubtedly oversimplified. The effective area described as being on a slope facing the apex could be on a sharp crest or might result from cross movements in the endolymph. It is undoubtedly finer in action than I have pictured it.

The curves of the propagation waves in the basilar membrane as described by various mathematicians and physicists vary some-

what, but all are characterized by a sharp break directed toward the apex.

The movements involved in hearing are undoubtedly very small and are to be reckoned in fractions of a micron.

If the thesis here presented is correct, then the area of stimulation has been narrowed from a broad zone of vibration in the basilar membrane to a narrow, small slope in the tectorial membrane. It is still not narrowed to a single line of cells in the organ of Corti, much less to a single cell, but it is likely that further selection and narrowing occur within the complex structure of the organ of Corti itself. Undoubtedly, for example, the phalangeal processes of Deiter's cells play a role, but whether they take part in the actual transmission of the stimulating impulse or merely act as braces against the pull of the fibrils, is open to conjecture. The pull of the fibrils at 30° to 40° with the long axis, would tend to tip the transversely oriented arches. The position of the phalangeal processes directed as they are, upward apical-ward and inward, would seem to be ideal to brace against such a pull by the fibrils.

## SUMMARY

A new concept of the translation of the broad sound vibrations in the cochlear fluids into a localized stimulus which is applied to the organ of Corti is presented. The mechanism involved consists in a shearing force applied through the tectorial membrane, which is not transmitted to the organ of Corti, because of the transverse flexibility of the tectorial membrane, except at one spot, located where the shearing force is directed in the axis of the relatively firmly fixed and non-flexible fibrils. A previous study has revealed that the tectorial membrane is inserted upon the organ of Corti, and together with two cell masses, one in the external sulcus and one in the internal, forms a flexible arch which is closely applied to the arch supporting the hair cells.

Each of 14 listed anatomical factors from the cochlear duct seem to fall naturally into a useful place in this concept.

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# XXX

# VENOUS EXTENSION OF A MIXED SALIVARY GLAND TUMOR

CASE REPORT

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For many years the so-called "mixed salivary gland tumors" have presented an intriguing and difficult problem to the conscientious surgeon. Although readily accessible and removable as a rule, these tumors have recurred after apparent complete removal. Such recurrences may have occurred soon after excision or after many years. Usually these recurrences are attended with an increasing cellularity of the tumor and occasionally by definite invasive characteristics. Quite rarely, actual metastases have been reported.[11]

McFarland,[9] who has perhaps collected the largest series of mixed salivary gland tumors, reported that out of 297 tumors, 60, or 23.23 per cent, recurred. He broke this down further in his report of 301 mixed tumors. Two hundred seventy eight, or 9.36 per cent, were of the parotid gland, 22, or 6.1 per cent, were of the submaxillary gland, and one, or 0.3 per cent, was of the sublingual gland. In recurrences he found that of the 278 tumors of the parotid, 20, or 21.5 per cent, recurred; of the 22 tumors of the submaxillary gland, 8, or 36 per cent, recurred; and the one tumor of the sublingual gland recurred. He found that the histological picture gave little aid in determining which cases were more likely to recur.

Such a pessimistic attitude is refuted somewhat by Maxwell and Buxton,[2] who studied 227 patients with primary tumors of the parotid gland. Twenty-seven patients in this series had one or more recurrences, an overall incidence of 13.3 per cent patient recurrences for tumors of all types of the parotid gland. In this series 110 patients had operative removal for benign mixed tumors with only 12 recurrences, or 10.9 per cent. Fifty-seven of these patients were less than five years postoperative and cannot be considered cured, as recurrences may occur as late as 20 or 30 years after excision.

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Read before Houston Ophthalmological and Otolaryngological Society, February 14, 1952.

Such statistics have led various investigators[5, 9, 12, 13] to speculate as to why recurrences developed in benign lesions of this type, many of which were quite superficial and readily accessible to excision. Histologically, the mixed salivary gland tumors have long troubled the pathologist. Some tumors show a characteristic picture of masses of epithelial cells often showing a glandular arrangement, mucoid connective tissue with mucin production, cartilage (probably a pseudocartilage), and scattered masses of lymphoid tissue. Much speculation as to their origin has been made. The English school leans more toward a bronchiogenetic origin with misplaced cell rests, giving rise to a teratological lesion. Ewing felt these tumors probably represented a metaplastic change in the epithelial cells of the glandular structures of the salivary gland or seromucous glands.

Usually these tumors are encapsulated, although they occasionally infiltrate the surrounding tissue. Naturally, where the capsule is thin or absent, removal is more difficult and recurrence more likely. Possible causes of recurrence may then be stated as (1) incomplete removal, (2) small separate growths in the capsule and inadvertently left behind, (3) breakage of tumor capsule and seeding at time of removal (these tumors grow readily "in vitro"[6]), (4) possible second neoplasm, (5) possible venous extension of tumor.

## CASE REPORT

Mrs. R. E. B., aged 64, a doctor's wife, complained of a hard swelling under the left jaw for two and one-half years. It had enlarged very slowly during this time to the size of a hazel nut. It was painless and did not fluctuate in size. Roentgenograms of the lesion did not reveal any calculus. Physical examination and laboratory studies were not significant.

On September 21, 1951, under pentothal sodium anesthesia, the tumor was excised. Little trouble was encountered in the removal of the gland until its duct was about to be ligated. At this time, it was noted that there appeared to be two separate ducts coursing forward under the mylohyoid muscle. Both were doubly ligated and the gland completely removed. On sectioning the gland a yellow tumor, not well encapsulated, was seen occupying most of the gland. The patient did well and was discharged on the third postoperative day. The wound healed by primary intention.

Microscopic report stated:

"The tumor has some fibrosis separating it from the gland tissue but this separation is not complete; areas of tumor tissue with very

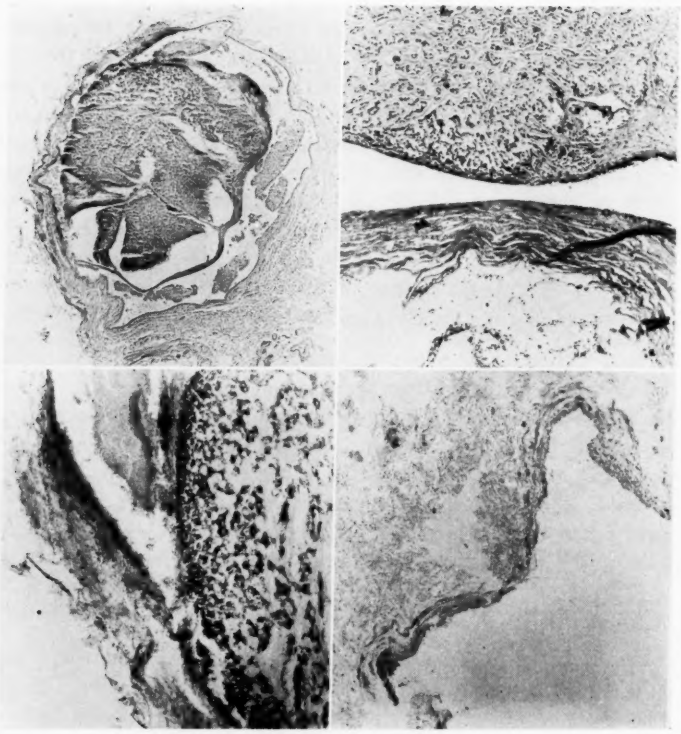

Fig. 1.—Low power showing mixed tumor nearly filling large vein near its exit from the submaxillary gland. Note blood between tumor and venous wall.

Fig. 2.—Higher power showing portion of mixed tumor with its typical histological arrangement of masses of epithelial cells, the mucoid connective tissue with mucin production and the strands of fibrous connective tissue. Note especially the intima lining the inner wall of vein supported by fibrous connective tissue.

Fig. 3.—Highest power of Figure 2. Note tumor attached to venous wall with small remaining lumen filled with blood. Again note intimal lining of vein.

Fig. 4.—Cross section of vein beyond tumor extension. Note typical venous wall in cross section.

small cells lie along side of normal tissue with only a thin loose fibrous area between.

*Tissue Diagnosis:* The tumor varies considerably in histology. All has some cartilage-like substance and very little myxomatoid material; through this stroma pass narrow groups of cells in a lobular pattern. In some areas cells are much more numerous with less stroma. A few areas of older growth are chiefly hyalinized fibrous tissue with small active tumor areas on the periphery.

The vein-like structure passes through normal glandular tissue and is completely filled with tumor but its distended wall is recognizable and in some areas where the tumor is detached from it, endothelial lining persists. In one of the smaller branches there is blood between tumor and wall; the wall here is infiltrated.

Elastic tissue stain confirms the impression that this is a vein. Some elastica is found around the ducts but is in much smaller amount and in less regular distribution.

The "cartilage-like material" is much less suggestive of cartilage in the section than it was in the gross. None of the small lacunae are found containing cartilage cells. The material is rather a homogeneous matrix within which are groups of cells. The rounded yellowish body is chiefly matrix with a few growing cells.

*Diagnosis:* Mixed tumor of submaxillary.

*Supplementary Report:* Because of the importance of proving beyond a doubt that this tumor is actually within the lumen of a vein, further sections were made.

Microscopically, this shows a normal vein with normal lining.

As one approaches the tumor inclusion, tumor appears first as a fragment lying free in the lumen surrounded by blood. As one passes backward, this tumor nodule increases in size and forms an attachment to the lining and wall of vein. Becoming larger it forms the distended mass which was seen in sections taken from the opposite side of this same block."

Dr. Stuart A. Wallace, of the Pathology Department of Baylor University College of Medicine, kindly reviewed the microscopic sections and concurred that mixed salivary gland tumor was actually growing in the lumen of the vein.

## COMMENT

One might well wonder how often a mixed tumor has invaded some vein had the exact serial section been taken. In this case, the vein was distended with tumor to the point that it resembled the gland duct. Fortunately, the microscopic picture showed that excision had been well beyond the tumor. Further, in as much as the whole gland was removed along with the tumor, the chances for a cure are brighter. The British school, and more recently the West Coast Group, have advocated parotidectomy, removing the gland along with the tumor to insure against leaving any neoplasm.

## CONCLUSION

A case of mixed salivary gland tumor of the submaxillary gland is presented where the tumor has actually invaded a large vein. It is postulated that this may be a cause for some of the recurrences after surgical excision of these tumors.

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XXXI

# ANALYSIS OF HEARING LOSS PATTERNS IN A RURAL ILLINOIS SCHOOL SYSTEM

## A COMPARISON OF SPRING AND FALL AUDIOMETRIC THRESHOLDS

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The general patterns of hearing loss in a rural Illinois school population have been previously described.[1] The occurrence of hearing losses at specific frequencies has also been set forth.[2] These previous studies may be briefly summarized by restating some of the principal conclusions: (1) The statistical analysis of these studies showed that hearing losses of 30 decibels or more occurred in 15.29% of the boys and 10.10% of the girls in the rural population tested. The analysis further showed that the indicated incidence of hearing loss is one which would occur by chance less than one time in 100. This difference is therefore justified as being of high significance statistically. (2) Hearing impairment in the studied population occurred more often in the right than in the left ear. (3) Boys showed a greater tendency than girls for high tone loss; this specific finding agrees with previous results in samples of urban students of the same age.

The present material covers the group of students who were identified as having a hearing loss at the time of the spring hearing survey. This survey was conducted in the months of March and April. The students with reduced audition have been studied to determine any statistically significant differences between the original spring audiogram and a retest audiogram made in the fall of the same year (September and October).

This analysis covers all the children in grades 5, 7 and 9 in two Central Illinois counties, Dewitt and Piatt. The 6062 students were regarded as a rural population by the Public Health Physician who was responsible for this survey of hearing acuity in his Public Health Unit. The pupils of the indicated grades were given a pure fre-

From the University of Illinois.

quency sweep test of 15 decibels intensity at seven frequencies (256-11,584). All pupils who did not pass this sweep test were given an individual pure frequency air conduction audiometric test on the same seven listed frequencies. Each child who had a hearing loss of 30 decibels or more in either ear at any one or combination of the seven test frequencies was referred by the health unit authorities to his individual family physician.

This present study is immediately concerned with the test-retest program on those children who were shown to have a 30 decibel loss at the time of the spring test. The parents of all the children shown to have a 30 decibel or greater loss were to take their children to their family physician for medical examination and to avail themselves of such treatment as would be suggested. The school nurses of the Public Health Unit visited individually the homes of the indicated children and explained the importance of early medical advice and treatment following the audiometric survey.

As might be expected, not all parents took their children to the family physician. This report is a statistical study of those children who did not receive medical treatment. Those individuals with an indicated hearing loss in the spring were retested in the fall of the same year; the change in audiometric thresholds is the measure with which we are here concerned.

The threshold value in decibels for each specific frequency or frequencies which had caused the individual to be included in the "hearing loss" category was made the basis of the present analysis. The spring threshold value was compared to the fall retest value for each frequency, for each individual. Audiograms on all seven frequencies were taken in the spring and fall, but for this study only the values of 30 decibel or greater loss were studied. This analysis therefore is not a general study of the shift in audiometric thresholds for a complete audiogram but rather a study of those thresholds which had caused the individual to be included in the hearing loss category.

The threshold measurements were obtained with a pure frequency audiometer by two testers who had extensive audiometric training and experience. A test-retest study on a small portion of the sample indicated for both spring and fall that the obtained results were highly reliable at the time of each test.

Four particular results will be considered for this group of individuals which did not receive medical treatment:

TABLE 1.—AN ANALYSIS OF VARIANCE IN THE IMPROVEMENT OF HEARING THRESHOLDS IN BOYS AND GIRLS AT SIX TEST FREQUENCES.

| SOURCE OF VARIANCE | SUM OF SQUARES | DF | VARIANCE | F |
|---|---|---|---|---|
| Between | 8,676.9 | 11 | 788.8 | 2.133 |
| Sexes | 3,019.3 | 1 | 3,019.3 | 8.166 |
| Frequency | 4,297.2 | 5 | 859.4 | 2.324 |
| Interaction | 1,360.4 | 5 | 272.1 | |
| Within | 137,920.2 | 373 | 369.8 | |
| Total | 146,597.1 | 384 | | |

F: Sexes/Within
F, 1 & 373: 0.1%, 10.83; 1.0%, 6.64; 5%, 3.84

F: Frequency/Within
F, 5 & 373: 0.1%, 4.10; 1.0%, 3.02; 5%, 2.21

1. In this study, the girls showed greater improvement than did the boys. An analysis of variance was done to test the statistical significance of changes in threshold values between spring and fall tests when all cases and all frequencies were considered together. The values of the analysis are shown in Table 1. For those boys and girls whose thresholds were poorer in the fall there was no statistical difference in the extent of the decreased threshold. About 22% of both sexes showed such a decrease. On the other hand, about 78% of both sexes showed a distinct improvement in the audiometric threshold between the two tests. The girls showed improvement of 27.1 decibels compared to 18.7 decibels for boys. This difference in improvement is significant at the 1% level of confidence.

2. In this study, the right and left ears showed equivalent patterns of improvement. In the light of previous findings regarding differences in incidence of hearing loss in the two ears, the subjects of the present study were examined to ascertain if different patterns of improvement might exist. Six analyses of variance, one for each frequency from 256 cycles to 8192 cycles, were carried out to compare at each frequency the improvement in right and left ears between spring and fall thresholds. These six analyses can be summarized by stating that no statistically significant difference was

TABLE 2.—CHANGES IN AUDITORY THRESHOLDS OF UNTREATED MALES.

| | FREQUENCY | | | | | | |
|---|---|---|---|---|---|---|---|
| | 256 | 512 | 1024 | 2048 | 4096 | 8192 | AVE. |
| Boys showing improved acuity | | | | | | | |
| Spring Threshold | 35.0 | 31.5 | 33.2 | 41.8 | 43.2 | 47.9 | 39.6 |
| Fall Threshold | 13.4 | 15.0 | 17.8 | 18.6 | 21.1 | 29.7 | 20.9 |
| Improvement in Threshold | 21.6 | 16.5 | 15.3 | 23.2 | 22.0 | 18.2 | 18.7 |
| Boys showing decreased acuity | | | | | | | |
| Spring Threshold | 30.0 | 11.7 | 15.0 | 47.5 | 31.9 | 15.3 | 21.7 |
| Fall Threshold | 41.7 | 36.7 | 32.5 | 57.5 | 46.2 | 32.8 | 38.3 |
| Decrease in Threshold | 11.7 | 25.0 | 17.5 | 10.0 | 14.4 | 17.5 | 16.6 |

found for either ear of either sex at any one of the six frequencies tested. The thresholds for all frequencies tended to improve, but no differential improvement in ears for either sex was demonstrated for any specific frequency.

3. The thresholds for low frequencies tended to improve more than the thresholds for high frequencies. "t" scores were computed for the size of the significant difference between spring and fall thresholds at each frequency (256-8192). A general trend for the frequencies 256 through 1024 to improve more than the higher frequencies was noted. Nine of the 12 differences 256-1024 were significant at the 1% level of confidence. Above 1024 cycles this trend for improvement was much less significant statistically; only four of the 12 "t" values were significant. This pattern is essentially similar for each sex. Up through 1024, four of six "t" values are significant in boys, whereas five of six values are significant for girls. Above 1024 cycles, two of six differences are significant for each sex. (See values, Tables 2 and 3.)

4. The thresholds were better in the fall than at the time of the spring test. This improvement of thresholds might be said to be the expected pattern as suggested by Guild, Polvogt and Sandstead.[3] This present finding reaffirms the previous observation of this author[4]

TABLE 3.—CHANGES IN AUDITORY THRESHOLDS OF UNTREATED FEMALES.

| | FREQUENCY | | | | | | |
|---|---|---|---|---|---|---|---|
| | 256 | 512 | 1024 | 2048 | 4096 | 8192 | AVE. |
| Girls showing improved acuity | | | | | | | |
| Spring Threshold | 40.7 | 33.8 | 43.9 | 20.0 | 38.0 | 42.7 | 41.3 |
| Fall Threshold | 13.9 | 14.4 | 14.3 | 9.4 | 13.8 | 16.7 | 14.2 |
| Improvement in Threshold | 26.8 | 25.2 | 29.6 | 28.7 | 27.9 | 26.0 | 27.1 |
| Girls showing decreased acuity | | | | | | | |
| Spring Threshold | 21.3 | 26.3 | 17.0 | 38.1 | 41.7 | 62.5 | 27.9 |
| Fall Threshold | 33.8 | 37.5 | 35.0 | 37.5 | 60.0 | 77.5 | 42.3 |
| Decrease in Threshold | 12.5 | 11.3 | 18.0 | 17.5 | 22.0 | 15.0 | 14.4 |

that it is preferable to conduct a hearing survey in the fall because of this indicated improvement in the thresholds of individuals with hearing loss.

It should be pointed out that the percentage of occurrence of hearing loss as computed for this population was determined from the fall audiograms. The general figures of 10% for girls and 15% for boys remain. The tests on this particular population took place in the early spring and fall of 1949. It was the opinion of the Public Health physician that the winter and spring of 1948-1949 was a particularly "bad" year for respiratory and ear difficulties in the population of these two counties.

## SUMMARY

This study has concerned itself with changes between spring and fall audiometric thresholds in a group of rural Illinois 5th, 7th and 9th grade children. The measured changes in thresholds have been studied by an analysis of variance technique and the following conclusions highly justified statistically for the population under test:

1. Girls showed a general tendency to improve more than did the boys.

2. Although all frequencies tended to show improvement in the thresholds there was no significant difference between the left and right ears for either sex.

3. The frequencies up to 1024 cycles tended to improve more than the thresholds for frequencies above 1024.

4. It would appear better to do the hearing survey in the fall than in the spring. Seventy eight per cent of the impaired thresholds showed a significant improvement in the fall test.

5. In audiometric studies the results may be confused if all audiogram results are lumped together and general averages computed. This author would feel that the present study indicates that more meaningful results will be obtained if the thresholds are considered by sex groups and specific frequencies.

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XXXII

# LINGUAL THYROID: WITH REPORT OF A CASE

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Of sufficient rarity to warrant the reporting of even a single case is the condition of lingual thyroid, sometimes called lingual goitre, or simply aberrant thyroid. When one recalls the various anomalies of position which this gland may seek, the base of the tongue, various way stations along the thyroglossal duct, the substernal region, the thorax, and rarely the nasopharynx, it becomes clear that as otolaryngologists, we must know something of the structures and function of this gland in health and in disease.

The incidence of lingual thyroid is reported to be about one in 2500 to 4000 cases of all types of thyroid disease. It is said to be more common in the female. The case which we are reporting is that of a 30-year-old male. The condition may make its appearance at any age, commonly in the second and third decades. One of the earliest reported cases (Heckman, in 1869) was that of a newborn who died 16 hours after birth due to asphyxiation produced by a thyroid tumor at the base of the tongue.

When thyroid tissue resides at the base of the tongue, it is as a rule all, or nearly all, of the thyroid tissue possessed by the patient. It may be normally functioning thyroid, or it may be a gland, the seat of any of the diseased states which may affect the gland in its usual position. This includes tumors, adenoma and carcinoma.

The condition is usually discovered quite unexpectedly during the examination of the patient who presents himself for treatment of mild sore throat or spasmodic coughing attacks if the lesion is small. Occasionally the lesion may have attained sufficient size to produce dysphagia or dyspnea and associated signs of respiratory distress. In some cases, sudden and rather profuse bleeding from the vessels coursing over the surface of the tumor may be the first symptom.

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The appearance of the lesion, best seen when the tongue is drawn forward, is that of a smooth, circumscribed swelling, bluish-red in color and confined to the area rhonbica of the tongue. Its consistency is soft and one has the impression of an encapsulated tumor. The conditions requiring differentiation are: (1) simple cysts of the lingual tonsil, (2) mixed tumor of the base of the tongue, and (3) some types of angiomata. A lymphoma may also appear strikingly similar; but as a rule, a lymphoma having attained any size at all will be accompanied by glands in the neck.

The diagnosis may be suspected on clinical examination, especially if the thyroid gland in the normal position cannot be palpated. Biopsy of the lesion would help to settle the diagnosis if other methods are not available. But the development of radioactive isotopes in medicine has, as one of its most practical applications, given us considerable help with problems of this type. By means of radioactive iodine ($I_{131}$) the diagnosis is rendered fairly certain without first recourse to biopsy or excision of the mass.

A tracer dose of radioactive iodine ($_{131}$) is administered in a dosage of 20 microcuries in 250 cc of water in the form of "cocktail" which the patient drinks. Forty-eight hours later a Geiger counter is pointed towards the various regions under consideration, as shown in Figure 2. The counts obtained over each site are tabulated and the total uptake of Radioactive Iodine estimated. Figure 1 in the diagram indicates the usual position of the isthmus of the thyroid gland; Figures 2 and 3 the right and left lobes of the gland; Figure 4 the region of a pyramidal lobe if present; and Figures 5, 6 and 7 the suprahyoid region and the base of the tongue. The Geiger counter could, of course, be applied to any number of thyroid tissue in a dermoid cyst. The counts per minute which are registered when the counter is directed to each of several sites are then compared, and the preponderance of thyroid tissue will reside in those regions showing the higher counts. Allowance is made for "scatter" when comparing sites which are in close proximity.

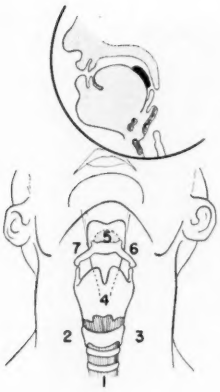
## REPORT OF A CASE

Mr. R. L., aged 30, was first seen four weeks following a rather severe sore throat associated with spasmodic coughing which caused him to be alarmed. The patient had rarely been the subject of sore throats in the past. He had no difficulty in swallowing either foods or liquids and there was no respiratory discomfort, apart from coughing. He had not had fever or malaise. By the time he first appeared for examination he was symptom free. Examination showed a mass

Fig. 1.

at the base of the tongue about the size of half an English walnut appearing as described above. It was covered by mucous membrane continuous with that of the tongue and beneath which were coursing several large veins. There was no evidence of inflammation in the throat. Mirror examination showed the tumor to be well clear of the valleculae when the tongue was protruded maximally. The tonsils were present but not diseased. The mass felt only slightly firm to the touch. There were no palpable lymph glands in the neck. Generally, the patient showed none of the signs of thyroid dysfunction, and the basal metabolic rate was minus 5. There was no palpable thyroid tissue in the usual sites of the gland. A clinical diagnosis of Lingual Thyroid was made. The appearance of the lesion is illustrated in Figure 1.

The patient was given a tracer dose of 20 microcuries of radioactive iodine and the 48 hour uptake was estimated to be 37.5%, well within normal limits. The results of the directional counts are shown in the table accompanying Figure 2. It will be noted that the counts per minute obtained when the Geiger counter was pointed toward sites 1, 2, 3 and 4 were comparatively low. In contrast are the very high figures obtained when the counter was directed to the regions 5, 6 and 7. Allowing for "scatter" which could explain the counts over sites 1, 2, 3 and 4, the inference is that the tumor at the base of the tongue is thyroid. Moreover, it probably represents practically all the thyroid tissue which this patient has.




*48 HOUR DIRECTIONAL COUNTING*

| SITE | NET COUNTS/MIN. |
|---|---|
| 1 | 62 |
| 2 | 84 |
| 3 | 205 |
| 4 | 280 |
| 5 | 3277 |
| 6 | 7097 |
| 7 | 6307 |

Fig. 2.

## TREATMENT

A decision must be made as to whether the patient stands to benefit by surgical excision of the lesion, a therapeutic dose of Radioactive Iodine or nothing at all, save observation from time to time. The aberrant site of the thyroid gland is not inconsistent with normal function. Carcinoma occurring in a lingual thyroid has been reported, but its incidence here appears to be no greater than when the gland occupies its usual position in the neck. In this connection, it is well to remember that the Radioactive Iodine test alone will not rule out a carcinoma, for well-differentiated carcinoma of the thyroid may take up relatively normal amounts of the isotope.

If a thyroid gland, normal except for its aberrant location, is removed in toto (as it should be if done at all) a myedematous state is likely to develop and the patient required to have replacement therapy the rest of his days under medical supervision. Although such replacement therapy is adequate, we do not feel that surgery should be resorted to merely because the thyroid gland happens to reside at the base of the tongue instead of in its usual position in the neck. In the case reported, the symptoms were so few and transient that for the moment we decided to withhold surgery. If the tumor enlarges, causing dysphagia, respiratory distress or bleeding, surgery should then be employed.

It is felt that once surgery is decided upon it should be with a view to complete removal of the tumor and not partial excision undertaken with an idea of preserving some functional thyroid tissue.

The reason for complete removal is that regeneration and reappearance of the mass is entirely possible otherwise. Then, of course, the probabilities would be in favor of having to employ replacement therapy. Several writers, in describing surgical removal of a lingual thyroid, mention the postoperative occurrence of tetany. This would seem hard to justify, we feel, on purely embryologic grounds, for the parathyroid glands develop quite independently of the thyroid. The presence of lingual thyroid should not in any way interfere with normal development and position of the parathyroids.

If surgical excision of the gland is carried out, it may be done very easily through the mouth, using traction sutures to assist in drawing the tongue forward and out of the mouth. There is no need, unless the tumor is quite large, to employ the procedures of lateral or transhyoid pharyngotomy. Provided of course that ether is not used as an anesthetic, electrosurgery makes bleeding less formidable.

If such a lingual thyroid, which has had diagnostic radioactive iodine in a tracer dose, is removed surgically, preferably near the time of the estimation of the uptake of RAI, then the entire specimen should be submitted to examination by radioautographs. These allow a much more accurate estimation of the uptake of the isotope by the thyroid and also indicate, in some cases, where the sections for microscopic examination should be taken. In this way a possible malignancy of the thyroid may be revealed while there is yet time to treat it with at least some hope of success.

SUMMARY

1. The condition of lingual thyroid is described. The clinical features of the diagnosis are listed.

2. The use of a tracer dose of radioactive iodine to help in establishing the diagnosis is outlined.

3. A case of lingual thyroid in a 30-year-old male is given together with the results of a therapeutic test of radioactive iodine.

4. The treatment is discussed. It is recommended to leave a normal lingual thyroid in situ if it isn't causing the patient more than occasional discomfort. If surgical excision is necessary, it should be complete removal followed by replacement therapy should myedema develop.

5. The importance of complete histological studies of specimens of thyroid removed is stressed together with radioautographs if a therapeutic dose of radioactive iodine precedes the surgery.

243 CHARLES STREET.

# XXXIII

# SCLEROMA

## REPORT OF A CASE APPARENTLY CURED BY STREPTOMYCIN

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Scleroma is a benign, proliferating, chronic granulomatous disease primarily localized in the mucous membrane of the upper respiratory tract. The first reports described the nasal lesions and it was called rhinoscleroma, but after it was shown that the disease occurred in other locations, the more inclusive term scleroma was chosen at the Second International Congress of Otorhinolaryngology in 1932.[25] The condition initially was described by von Hebra and Kaposi in 1870.

The first cases of scleroma were seen in Europe and endemic foci were found in Galicia and the Pripet marshes. All of the cases seen in this country were in persons of foreign birth until Watkins in 1921 reported the disease in an American-born negro. Since then, endemic areas have been discovered in South Carolina and southern California in this country, portions of India, South America and the Balkan states.

Miller[20] reports the disease as occurring almost equally in both sexes, but Hanstedt[13] and Reyes[28] believe that it predominates in women. Sivak[29] believes that women show a greater affinity for the hyperplastic forms of the disease and that scleroma is prevalent in regions with an abundance of fresh water. No age group is immune, but the greatest incidence is between 15 and 35 years of age. One hundred twenty cases have been reported from the United States and Canada and, of these, 26 were native-born. This report adds an additional native born case (Table 1).

The condition is not inherited. Reyes[28] believes that the disease is not contagious and is contracted through poor personal hygiene. In this he is supported by Eisenstodt[6] and Miller[20] Hara[14] and his co-workers believe that scleroma is contagious and transmissable by

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TABLE 1.

CASES OF RHINOSCLEROMA REPORTED IN THE UNITED STATES.

| AUTHOR | | AUTHOR'S CASES | | TOTAL IN LITERATURE | |
|---|---|---|---|---|---|
| | | NATIVE BORN | FOREIGN BORN | NATIVE BORN | FOREIGN BORN |
| Goldstein | 1941 | 0 | 1 | 8 | 58 |
| Cunning | 1942 | 1 | 2 | 16 | 86 |
| Duane | 1942 | 0 | 1 | | |
| Dill | 1943 | 0 | 1 | | |
| Hara, et al | 1947 | 7 | 0 | 16 | 86 |
| New, et al | 1948 | 0 | 1 | | |
| Putney | 1948 | 0 | 1 | | |
| Miller, C. K. | 1948 | 1 | 0 | | |
| Som | 1949 | 0 | 1 | | |
| Miller, A. H. | 1949 | 2 | 1 | | |
| Morwitz, et al | 1951 | 0 | 1 | | |
| Eisenstodt | 1951 | 0 | 1 | | |
| Titche | 1952 | 1 | 0 | 26 | 94 |

long contact under poor hygienic environment, and Sivak[29] points out that, epidemiologically, the appearance of scleroma in families is of importance.

Von Frisch in 1882 first described a bacillus which he believed to be the causative agent. The organism is a gram-negative bacillus, usually assuming the shape of plump rods with well-rounded ends. The organisms are mostly intracellular within the foam-cells, but may be seen extracellularly after rupture of the cells. Som and Jaffin[30] state that it may be a strain of the Bacillus mucosus capsulatus. Since the disease has not been able to be reproduced in lower animals by this organism, many have not believed it to be the causative factor. Cunning and Guerry[2] and Figi and Thompson[8] thought that it was a secondary invader, while Miller[21] believed that, if it was the etiologic factor, it probably functioned in symbiosis with some other agent. On the other hand, Levine and Hoyt[18] describe a characteristic culture pattern for Klebsiella rhinoscleromatis and could not find the organism in the nose or throat of 267 normal persons.

Gerber[9] recognized the lesion as a chronic inflammatory process accompanied by perivascular cellular infiltration and connective tissue proliferation. Reyes[28] described the pathology of scleroma as being a tumor formation of varying volume, of waxy or reddish appearance and with a decided tendency to ulcerate and spread. Dill[4] and Hara et al[14] describe three stages of the disease: the edematous or catarrhal stage, which resembles any chronic inflammatory reaction and during which the diagnosis cannot definitely be made; a second or granulomatous stage which produces an increase in the size of the lesion and is characterized by the large vacuolated cells of Mikulicz and masses of hyaline tissue called Unna's bodies; and the final stage of sclerosis and deforming scar formation. Hanstedt[13] divides the first stage into an initial or atrophic one in which the clinical picture is that of atrophic rhinitis, and an indifferent granulation second stage in which round cells and plasma cells occur.

Crawford and Gerundo[1] found the stage of sclerosis predominant in one region of the respiratory tract with newer lesions present in others. These authors found a striking similarity between this disease and leprosy of the skin, especially in the fact that the organisms of both diseases are found in the same types of cells, as the Mikulicz and lepra cells are derived from the reticuloendothelium.

Scleroma begins in the anterior part of the nose and slowly makes its way posteriorly to the nasopharynx, though the nose may remain the only site of involvement. During the second stage, the upper lip may be invaded and produce external deformity. The pharynx may be involved, but usually is by-passed and the disease involves the larynx and sometimes the trachea and bronchi. The onset is insidious and the course and severity are very variable. Spontaneous arrest even may occur. There is no pain, fever or general constitutional symptoms, except those due to secondary infection or to the respiratory obstruction.

Conditions from which scleroma has to be differentiated are nasal diphtheria, syphilis, tuberculosis, lupus, leprosy and atrophic rhinitis. In its early stages, scleroma resembles atrophic rhinitis, since both conditions present a foul, odorous, purulent discharge with crusting, but in scleroma, removal of the crusts reveals narrowed nares and a firm red mucosa. Jaffe[15] states that whether or not there is a relation to atrophic rhinitis and ozena has not been established yet and Hanstedt[13] believes that the differentiation cannot be made in the early stages.

A positive diagnosis can be made by finding the organism with its characteristic cultural pattern, by a positive biopsy and by a positive complement-fixation test. Mohanty[22] believes that biopsy is positive only in the third stage of the disease and therefore is of no value earlier. Levine and Hoyt[16] and Wexler[32] stress the value of the bacteriological findings and the serological test, but Cunning and Guerry[2] state that the diagnosis can be established only by biopsy.

Many and various modes of treatment have been employed. Surgical excision of the lesions sometimes followed by roentgen irradiation or radium has been used without too much success but Dixon,[5] Dill,[4] Crawford and Gerundo[1] believed irradiation was of assistance by producing fibrosis. Rao and Menan[27] noted some improvement in the local condition with injection of urea-stibamine. Goldstein[11] reported a decrease in the lesions in one case following a combination of radiation therapy and an autogenous vaccine, and Ghosh and Panja[10] confirmed this in one case. Farhes, Barrat and Berge[7] employed electrocautery and electrocoagulation to remove the lesions and no recurrence was noted. Eisenstodt[6] found the disease to become dormant after electrocoagulation of a portion of the lesion and the administration of potassium iodide. Sulfanilamide and sulfadiazine were tried by Cunning and Guerry,[2] and sulfathiazole by Jaffe,[15] without effect except to control the secondary infection, though Reyes[28] believed that roentgen rays and azosulfamide were the only effective therapeutic agents. Guerrier[12] obtained negative bacteriological and biopsy examinations with penicillin and roentgen therapy.

Streptomycin was used first by Hara and his co-workers,[14] and they could not recover the causative organism after six days in one case, and in another obtained marked reduction in the number of organisms. Quevedo[26] reported in 1947 that "although streptomycin is not the definite answer to the challenge of the disease, it is to date the best weapon that can be used against scleroma." Apparent cures with this drug have been reported by New,[24] Som and Jaffin,[30] Miller[20] and Curkovic.[3] Lewis and Ranger[19] obtained improvement with streptomycin after no effect had been obtained with deep x-ray therapy. Morwitz and Horwitz[23] have used dihydrostreptomycin with good results. The dose of streptomycin has varied from 0.5 gm every four hours to 1.0 gm a day, with a total dosage from 10 gm to 173.5 gm administered in from ten days to 74 days. Tan and Simuangco[31] stated that streptomycin produced sudden and dramatic improvement in those cases with laryngeal, tracheal and bronchial lesions.

REPORT OF A CASE

O. N. M., white male, aged 26, who was born in Texas was admitted to this hospital with a chief complaint of choking spells and difficulty in talking. He stated that in 1945 while in the Navy he noticed increasing hoarseness and was admitted to a Naval Hospital in Okinawa. A diagnosis of rhinoscleroma was made there, but a search of the records did not reveal that this was confirmed by finding the specific organism or by biopsy. No specific treatment was given at this time, and he was discharged a short time later. Since then he has had several attacks of choking with loss of consciousness, and he was brought to the hospital by his private physician for the performance of a tracheotomy.

Examination revealed both frontal sinuses to be clear on transillumination but the maxillary sinuses were dark and subsequent x-ray examination revealed a very marked thickening of the membrane of these sinuses. The nasal septum and the turbinates were covered with firm crusts, which on removal revealed slightly injected mucous membrane covered with small white spherical bodies. The soft palate, the tonsillar pillars and the posterior pharyngeal wall showed numerous small ecchymotic areas. The nasopharynx did not reveal any abnormality. On indirect laryngoscopy the vocal cords were slightly thickened and below the level of the cords there was a greyish mass.

The remainder of the physical examinations did not reveal any abnormality. Two days later direct laryngoscopy was performed and revealed no pathology in the larynx, but after a bronchoscope was inserted the posterior wall of the trachea in its upper two-thirds was found to be covered by a marked amount of roughened tissue which bled easily on manipulation. Portions of this tissue were removed with biopsy forceps and sent to the laboratory for examination. This was reported by the pathologist as acute necrotising inflammation, mucosa of trachea, type undetermined. Culture of this tissue and of the sputum did not reveal K. rhinoscleromatis. The patient's condition seemed improved after no specific therapy, and since he wished to return home for personal reasons, he was discharged with instructions to return in six weeks for examination.

The patient next was seen on July 2, 1951. He stated that he had not had any further attacks of choking but said that he noticed that he was expectorating crusts from his nose and throat, especially in the mornings. Examination revealed the same amount of crusting in the nasal chambers as had been noted previously, and indirect

TABLE 2.

ANTIBIOTIC SENSITIVITY

| | Antibiotic | | | | | |
|---|---|---|---|---|---|---|
| | Aureomycin | Bacitracin | Chloromycetin | Streptomycin | Penicillin | Terramycin |
| Concentration at which growth inhibited | 30 mcgm | 2 units | 30 mcgm | 5 mcgm | 10 units | 60 mcgm |

laryngoscopy did not show any pathology in the larynx or upper portion of the trachea. He was requested to return in six weeks for examination.

On August 14, 1951, the nasal chambers and larynx appeared unchanged. The crusts in the nose were sent to the laboratory for culture and were later reported as being positive for K. rhinoscleromatis.

The patient was readmitted to the hospital on September 27, 1951. Bronchoscopy was performed the following day and revealed some redness and roughening of the posterior portion of the larynx and of the posterior wall of the trachea down to the carina. The laboratory reported the antibiotic sensitivity of the organism isolated as shown in Table 2.

On October 4, 1951 streptomycin was begun in a dosage of 0.5 gm twice daily and given for 65 days, the patient receiving 65 gm of the drug. His symptoms rapidly abated and the nasal crusts cleared up markedly. Laryngoscopy and bronchoscopy were performed on December 11, 1951 and, other than for some slight scarring of the posterior wall of the trachea, no abnormalities or crusting were noted in the larynx or tracheobronchial tree. The patient was allowed to leave the hospital on December 14, 1951 and has been observed at frequent intervals since then. When he was seen last on March 3, 1952 he did not complain of any crusting or choking, and examination did not reveal any crusting or recurrence of the lesions.

VETERANS ADMINISTRATION HOSPITAL.

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# XXXIV

# IODISM

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Iodine is of importance physiologically because it is an essential constituent of the thyroid hormone. Therapeutically, it has long filled an important place in the physician's armamentarium. Of late years, it has also assumed a significant role in diagnostic roentgenology. The use of iodized oil in a bronchoscopic study with bizarre and fatal result led to the investigation of the deleterious effects of this drug, which forms the basis for this presentation.

Iodine is not found in nature in the free form; its salts, however, are distributed widely, although irregularly and sparsely. The metabolism and the effects of ingested iodine are said to depend both upon the form and the amount in which it is ingested and upon the state of the organism.

Iodides enter the body by various routes—ingestion, injection, inhalation, absorption from various orifices and cavities after introduction for therapeutic or diagnostic purposes, and also absorption through the pores of the broken or intact skin. After absorption, a process not yet fully understood, they are distributed uniformly throughout the extracellular fluid in the same way as are chloride and thiocyanate, rapidly reaching all the tissues and structures of the body. They make their appearance quickly in the milk and the saliva. In the blood they appear chiefly in the plasma, with only small amounts present in the blood corpuscles.

Normally, the thyroid gland is the chief repository for iodides. A rapid increase in the iodide content of this gland takes place with any increase in iodide intake. Apparently, the follicle cells of the thyroid gland seem to be the only tissue in the body having a specific affinity for iodide. This ion evidently acts only as a source of iodine for the manufacture of thyroid hormone. It is said that while the capacity of the thyroid is definitely limited, this gland can, on a low iodine intake, concentrate the element to more than ten thousand times the blood level.[1] In other parts of the body, one part per million is the amount normally present.[2] Iodides, as do quinine and cer-

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Presented as a candidate's thesis to the Am. L. R. & O. Society.

tain other drugs,[2, 3] easily pass through the choroid plexus to reach the cerebral spinal fluid. They also permeate the placenta to reach the fetus in utero. They even reach the chambers of the eye by passing through the ciliary blood vessels of the iris.

Excretion takes place rapidly, mainly in the urine and also in the sweat, but not in the feces. In addition, iodides are present in the excretions and secretions of most, if not all, of the glands of the body. The infinitesimal amount normally excreted rapidly increases with increased intake. This increase is readily demonstrated in the mucous glands of the upper and lower portions of the respiratory tract, in the saliva, in the excretions and secretions of the gastrointestinal tract and the liver, and in the sweat from the coil glands and in the sebum from the sebaceous glands of the skin. That iodides are easily secreted with the milk from the mammary glands is evidenced by iodism in nursing infants whose mothers are taking iodides.[2]

Although difficult to determine accurately, the minimal daily requirement of iodine is estimated at between 15 and 40 micrograms. The need varies with caloric turnover, rate of growth and the presence of pregnancy. Deficiency of this element leads to thyroid hyperplasia and to simple goiter.

### THERAPEUTIC USES OF IODINE

The definite, though limited, ameliorating effect of iodine on hyperthyroidism usually causes a fall of about 15 per cent in the basal metabolic rate together with corresponding symptomatic improvement. Since the advent of the antithyroid drugs for preoperative therapy in thyroidectomy, iodine no longer suffices except in the mildest cases, but it offers valuable supplementary therapy. In very small amounts, it is effective in the prophylaxis of simple endemic goiter.

Iodine has long been used as an "alternative," as an expectorant, as an antiseptic, and as a fibrolytic agent. In arteriosclerosis and during convalescence from apoplexy, iodides were administered almost routinely a generation ago, but the concept of general "alternative" medication has lost favor in the light of recent scientific advances. They have long served as expectorants in asthma, chronic bronchitis, and the later stages of acute bronchitis, but are considered too irritating to be given in the presence of acute inflammation.

A powerful germicide, free elemental iodine is also an effective fungicide, a potent amebicide, and a moderately active virucide. Its great chemical activity and its ineffectiveness in combined form, to-

gether with its corrosive action on tissues, have limited its usefulness chiefly to disinfection of the skin.

In granulomatous diseases, such as syphilis, leprosy and blastomycosis, large doses of iodide ion appear to have a fibrolytic and healing effect. Many syphilologists recommend its use as an adjunct to bismuth and mercury in the treatment of syphilis, but this therapy is highly empiric. Its mechanism is not satisfactorily explained, for iodide neither kills the organism nor influences resistance in a measurable way. Its value in association with penicillin therapy is even less certain. The need for iodides, therefore, will naturally diminish as more powerful specific treatment becomes available. They have been superceded in the treatment of actinomycosis and sporotrichosis. Curiously enough, tuberculosis, in contrast to other granulomatous diseases, responds adversely to iodides, with exacerbation and spread of the disease process often taking place.

It appears that responsibility for all the systemic actions of iodine rests with the iodide ion. Ordinarily, for systemic iodine therapy the simple salts, sodium or potassium iodide, administered orally, are sufficient. The mechanism of the action of these iodine compounds used systemically is not clearly understood. The rapid absorption of certain inflammatory exudates and especially of the gummatous lesions of tertiary syphilis is the most definite result. Under adequate dosage, lesions of this type in bone, skin, brain or other organs diminish or disappear. The iodide ion is not germicidal. The iodides do not directly lower blood pressure. They may, however, by affecting the production of thyroxin, indirectly have an effect on metabolism.

### DIAGNOSTIC USES

In roentgen diagnosis iodized oils are injected as contrast mediums. They are especially useful in localizing bronchial and pulmonary lesions, in gynecology and in diagnosing tumors of the spinal cord. Various vegetable oils are used, and according to the method of iodination, the oil may contain iodine alone, or iodine and chlorine.

Several water-soluble organic iodine compounds, notably diodrast, are now available for intravenous urography and for ureteral retrograde pyelography. In the necessary dose, sodium iodide is too toxic for intravenous injections. These compounds may also be used for arteriography, venography and angiocardiography.

### IODISM

In the ordinary sense, iodides are not toxic. In some patients, however, there develops an idiosyncrasy to them which is commonly

known as iodism. Although this condition has had much study, it remains poorly understood. As previously stated, it is reported to be related to the quantity of iodide given and especially to the state of the patient. The form in which the element is administered appears to be of no influence, and iodism may or may not recur if iodine is given at a later time.

Gastrointestinal intolerance to iodides is apparently due to local irritation and is usually manifested by nausea or diarrhea or both. Anorexia, vomiting and intestinal cramps also may occur.

Commonly, with small doses, coryza and acne occur, and, singularly enough, they may disappear when the dosage is abruptly increased to 90 grains or more daily.[1] Rashes and swellings of various kinds, and even exfoliative dermatitis may follow external application of iodine.

In general, iodism is manifested by acneform, maculopapular, vegetative (including frambesiform, fungating and granulomatous), vesicular, or bullous eruptions; coryza, conjunctivitis, bronchial irritation, and general swelling and inflammation of the mucous membranes; vascular symptoms such as edema, including angioneurotic edema, erythema, purpura and erythema multiforme, which may affect the skin and mucous membranes; fever and nervous irritability.[1]

As early as 1854 O'Reilly[4] described bullous ioderma, and in 1877 purpuric lesions were first described by Fournier.[5] Adamkiewicz[6] demonstrated iodides in the contents of a bullous lesion in 1878.

In iodism, the mucous membranes may be stained brown. Shock and dehydration may occur, as well as asphyxiation or edema of the glottis.[7]

Hand[2] observed that the irritative effects of iodine ions passing through the various glandular structures produce the symptoms of iodism. He ascribed the early prodromal symptoms of nasal congestion, nausea, anorexia, rhinorrhea, lacrimation and conjunctival congestion to excretion of iodine through the glands of the respiratory and gastrointestinal tracts and also to the irritative effect on the mucous membranes after excretion. The acneform eruptions he attributed in part to the irritative effect of iodides on the sebaceous glands, but thought the indirect effect of the glands of internal secretion such as the thyroid also deserved consideration.

CAUTIONS AND CONTRAINDICATIONS

When used systemically, iodides should be restricted to oral administration. Acute and violent iodism, colloidoclastic shock and pulmonary edema are dangers attending the intravenous injection of sodium iodide which outweigh the doubtful advantages of this route of administration.

With regard to caution in the use of iodized oils, the Council on Pharmacy and Chemistry of the American Medical Association warns:

"It should be emphasized that the injection of iodized oils is essentially a surgical procedure, introducing a foreign and possibly irritant body, and involving more or less risk, which should be weighed against the presumptive advantages, in comparison with the relative advantages and disadvantages of other measures. The following cautions should be especially borne in mind:

"1. Oils that have aged and darkened beyond their original color should never be used.

"2. Subarachnoid injections should be avoided, at least until all other means of diagnosis have been exhausted.

"3. Intratracheal and intrapleural injections should be avoided in tuberculosis of the respiratory organs and also when restriction of respiratory area would be contraindicated.

"4. The injection pressure should be carefully controlled, so as not to lacerate the tissues.

"5. Intra-uterine injections should be made only under fluoroscopic observations.

"6. Iodized oil should not be used for renal pyelography, except in the form of emulsion; and the injection should be stopped if pain is felt.

"7. Intravascular injections with iodized oil appear too dangerous; the use of emulsions for this purpose requires further study."[8]

The Council adds that because of its iodine content, chloriodized oil (iodochlorol) should be employed with caution in patients with pulmonary tuberculosis, active or latent hyperthyroidism and possibly some patients with thyroid adenoma.

With regard to intravenous urography, the Council cautions:

" . . . The excretory urogram should be made by persons experienced with this method and during the entire procedure the patient should be watched for untoward reactions. Ocular, oral and intradermal tests to detect sensitivity to intravenously administered iodine compounds are not reliable since reactions are more often due to a direct vascular effect. . . . Side effects which may be encountered include flushing of the face and neck, urticaria, fall in blood pressure, nausea, vomiting, lacrimation, salivation, edema of the glottis, bouts of coughing, "tight feeling" or choking sensation and cyanosis. These symptoms usually disappear over varying periods of time but fatalities have occurred.

"The intravenous use of the drug is contraindicated in patients with severe liver disorders, nephritis and severe uremia, and it should be used with caution in cases of active tuberculosis and of hyperthyroidism."[8]

Goodman[9] in 1933 listed contraindications to the use of iodized oils in pulmonary diagnosis as active pulmonary tuberculosis, recent hemoptysis, decreased vital capacity, most cases of asthma, and cachectic and debilitated patients. He added that age is not necessarily a contraindication. Zavod,[10] in 1948, observed that most of the complications which arise occasionally during or following bronchography can be avoided by ascertaining that the patient is not hypersensitive to cocaine or its derivatives and that he does not suffer from acute pulmonary disease, advanced emphysema or other illness that contraindicates the procedure. Since in many cases the lipiodol is retained for many months in the lungs, this author studied 50 patients spirometrically before and after a diagnostic bronchogram. In 90 per cent there was loss of pulmonary function and decrease in some pulmonary volumina one hour after the bronchogram. The highest average loss was in the reserve air, and daily spirometric studies after the bronchogram indicated that the loss is recovered gradually and that return to the prebronchogram level occurs within a maximum of five days.

### TREATMENT

Iodism in the more severe forms requires immediate discontinuation of the drug. Recovery usually follows rapidly. In some cases, however, the condition may persist and even prove fatal.

The administration of large quantities of fluids (4 liters daily) and sodium chloride (5 to 10 gm daily) hastens elimination of iodide. Just as the iodide ion replaces chlorine compounds, causing the displaced chlorine to be excreted, so in reverse mechanism, increased intake of chlorine by mass action frees the iodine ions, which are then excreted. Similarly, bromine acts by replacing chlorine and iodine and vice versa. This action, therefore, forms the basis for the modern treatment of both iodism and bromism.[2]

## Review Of The Literature

### IODISM IN BRONCHOGRAPHY

Iodized oil as a contrast medium for studying the bronchial tree in human subjects was introduced by Sicard and Forestier[11] in 1922 in France. They used lipiodol, which contains 40 per cent iodine in poppyseed oil in organic combination. Various other vegetable oils, including sesame oil, rapeseed oil, olive oil and peanut oil, have since been halogenated with iodine, an iodine-chlorine combination, or bromine for use in bronchography. Today, bronchography is a common procedure which is relatively safe when proper technic is em-

ployed. Acute reactions, however, do occur and occasionally prove fatal.

Severe reactions reported in the literature have practically all been attributed to iodism, anaphylaxis, or mechanical overfilling or obstruction of bronchi. Classifying the reactions to the instillation of iodized poppyseed oil into three categories, namely, anaphylactic shock and allergies, iodism appearing 24 to 48 hours after its introduction, and pneumonia developing two to ten days after the oil is introduced, Fortner and Miles[12] recently observed that iodism is due to dissociation of the poppyseed oil and the iodine. They mentioned the fact, well known clinically, that patients who swallow iodized oil are more prone to symptoms of iodism than those in whom the instillation is restricted to the respiratory tract and gave as the reason the more rapid dissociation of the iodine and poppyseed oil in the gastrointestinal tract. In their case, in which the diagnosis was pulmonary tuberculosis and bronchiectasis with repeated hemorrhages, complete symptomatic relief followed pneumonectomy. Granulomatous lesions observed in the removed lung on pathologic examination were believed due to lipiodol repeatedly instilled preoperatively to find a clue to the cause of the recurring pulmonary hemorrhages.

A similar but more spectacular case was that of Brody,[13] which he described as focal lipid granulomatosis, discovered at operation one month following bronchography with the use of iodized poppyseed oil. Microscopically and roentgenologically, oil droplets were identified in the numerous sharply circumscribed, firm, ovid, gray-brown nodules scattered beneath the pleura and throughout the parenchyma of the involved lobes of the lung, which was obtained by pneumonectomy. The bronchi to the lobes in which the granulomatous reaction occurred were partially obstructed by a bronchial tumor.

In a case of bronchiectasis, similar findings were reported in 1949 by Storrs, McDonald and Good[14] from the Mayo Clinic, except that no obstructing bronchial lesion was present. They used iodochlorol, a halogenated (chloriodized) peanut oil containing 27 per cent of iodine and 7.5 per cent of chlorine in organic combination. At operation three weeks after bronchography with this medium a granulomatous process was found in the lung. The left lower lobe contained many small yellowish gray nodules, some lying just beneath the pleura, and the left upper lobe contained a similar large area. Biopsy of one of the nodules demonstrated lipoid granuloma on fresh frozen section. Following left lower lobectomy and lingulectomy, the patient required oxygen therapy for four days. Post-

operative roentgenograms showed a large quantity of residual iodized oil in both lungs, with a great reduction in the amount six months later.

On chemical analysis, tissue from the granulomatous region contained three hundred times as much iodine per gram of wet tissue as did tissue from other regions of the lung which appeared grossly normal. The gross and microscopic pathologic findings were those of lipoid granuloma.

These authors found only three similar cases reported in the literature in which histologic study of pulmonary tissue gave evidence of a pathologic process apparently caused by the presence in the lung of iodized oil. In 1934, Rabinovitch and Lederer[15] mentioned a case in which postmortem study disclosed pulmonary changes resembling those seen in lipoid pneumonia which were apparently caused by iodized poppyseed oil administered some time before death. The next year, Wright[16] reported a case of bronchogenic carcinoma in which one year after bronchography, rapeseed oil was demonstrated roentgenologically in the lung, where it had apparently been trapped by the bronchial tumor. On postmortem examination of the lung in this region, much oil in droplets was present in a kind of "cellular syncytium," which had replaced most of the pulmonary tissue in the lobe involved. This author was of the opinion that the prolonged presence of the iodized oil had caused this reaction. The third case was that of Brody,[13] previously mentioned.

Storrs, McDonald and Good[14] observed that while the development of a granulomatous process in response to iodized oil occurs rarely, lesser degrees of such reaction may occur oftener than has been realized. They noted that histologic studies of postmortem material[17] and animal experimentation[18-21] with various iodized oils indicate usually little pulmonary reaction to these substances, but that some investigators[22-24] reported finding masses of epithelioid cells which had destroyed the normal pulmonary architecture in some regions. These masses of epithelioid cells, apparently small in most instances, could in their opinion represent a mild degree of the same reaction as was present in their case. Noting that the formation of granuloma and necrosis in the lung in their case resembled that described by Pinkerton[19] as occurring in response to animal oils of high fatty acid content, they reasoned that "perhaps this patient's lung possessed a very high degree of the lipase activity, described by several French writers, and rapidly broke down a portion of the peanut oil, releasing irritating fatty acids which caused the necrosis and the formation of granuloma."

In 1944, Boldrey and Aird[25] concluded from experimental studies that iodized oil injected intrathecally is less irritating to the meninges than iodized poppyseed oil. In view of this observation, and the long-continued use of peanut oil at the Mayo Clinic with only this one proved reaction of this type, Storrs, McDonald and Good[14] concluded that peanut oil in all probability is as good a base for iodized oil as other oils currently in use. The three similar reactions they cited occurred after the use of other oils. In the fatal case of iodism here reported, iodochlorol was the contrast medium used.

Mahon,[26] in 1946, reported his case of suspected bronchiectasis with fatal reaction following intrabronchial instillation of lipiodol as the only one of the kind for which there was a protocol on file at the Army Institute of Pathology. In this instance, after instillation of 10 cc of lipiodol into the base of each lung, roentgen examination was completed with the patient stating that he felt well, but an hour and a quarter later he was pronounced dead. This author concluded that the patient had allergic sensitivity to some constituent of lipiodol, probably the iodine, and the immediate contact between the allergen and the tracheobronchial tree, which was prepared to act as a shock organ, resulted in a rapid and severe asthma-like reaction in which the production of thick mucus predominated, causing bronchial obstruction, massive pulmonary collapse and death from asphyxia, as demonstrated at autopsy.

Waldbott,[27] however, was of the opinion that the pathologic condition in Mahon's case was typical of allergic asthma, that death resulted from obstruction of the bronchi by mucous plugs, and that pre-existing asthma and bronchiectasis should be held solely responsible. He observed that when much mucus is present in the bronchial tree, injections into the bronchi of additional viscous material such as lipiodol may induce obstruction of small bronchi followed by atelectasis of pulmonary tissue and pneumonitis or, as in this case, massive collapse of the lung when a main bronchus is involved. There is too much crowding in the bronchi. He suggested that injection of lipiodol be avoided in a patient having much mucus in the bronchial tree, or that it be preceded by bronchoscopic aspiration of mucus. When an accident occurs in such a case, he advised immediate insertion of a bronchoscope into the bronchi and aspiration of the obstructing material.[28]

Upon inquiry, Mahon[26] was informed by Good in a personal communication that some 300 bronchograms representing approximately 200 individual patients are obtained annually in the Section on Roentgenology of the Mayo Clinic. The patients at that insti-

tution are not tested for iodine sensitivity prior to lipiodol bronchography unless there is a history of sensitivity to iodine. Of the half dozen or so mild reactions occurring each year in patients sensitive to iodine, all have been controlled without fatality. He classified the reactions observed there following the use of iodized oil as:

1. Swelling of the parotid and submaxillary glands, beginning shortly after instillation of the oil and subsiding within 24 to 48 hours. This is the most common reaction, is seen three to four times a year, and occurs in patients without allergic history.

2. Asthma, occurring about once a year, always in persons who show other signs of allergy.

3. Urticaria, beginning almost immediately, not occurring over once a year and "probably on an allergic basis."

4. Skin eruption, papular or pustular, occurring ten to 14 days after the instillation of the oil, similar to a drug eruption and not seeming to have an allergic basis.

In his review of the literature Mahon[26] noted that when White and Bayliss[29] in 1943 reported from Australia a nonfatal case of acute iodism following lipiodol injection, they could find in the literature reports of only eight cases of severe reactions, three of which were fatal. Two of these cases, one of which terminated fatally, were reported by Scadding,[30] who stated that these were the first serious accidents from iodine sensitivity in over 2,900 examinations in which lipiodol was used. Mahon[26] noted ten other instances of severe reaction recorded in the literature, in five of which the iodism proved fatal. One of these fatalities[31] occurred in a girl aged 13, known to be asthmatic; she became cyanotic with weak pulse, and death ensued 20 hours after instillation of lipiodol. At autopsy, the findings were considered typical of bronchial asthma. In another of these fatal cases, the patient died a few minutes after instillation of lipiodol,[32] but details are lacking as they are in the other three fatal cases.[33, 34] In the five nonfatal cases,[35, 36] the reactions were diagnosed as massive collapse of the lungs.

With regard to sensitivity to iodine, it is of interest that a previous instillation of iodized oil had been given without reaction in the case reported by White and Bayliss,[29] and when the patient was tested for sensitivity to iodine after subsidence of the acute condition, the reaction was negative. Likewise in the nonfatal case reported by Scadding[30] there was no reaction previously when iodized oil had been instilled; also the patient in this case had been taking iodide by mouth prior to the injection of lipiodol without exhibiting symp-

toms of sensitivity to iodine. This author observed that he had frequently encountered mild reactions prior to the two severe ones—usually transient coryza with lacrimation and possibly mild urticaria or an erythematous rash, all lasting but a few days; less frequently, rashes more severe and of an acute irritant and urticarial type, occurring seven to ten days after injection; and headaches occurring rarely.

Noting previous reports of iododerma and iodism from iodized oil by Firth[37] and Carmichael,[38] and finding no record of a fatality from iododerma in the literature, Goldsmith[39] in 1936 reported a fatal case of iododerma following injection of 20 cc of lipiodol into the lung for diagnostic purposes. There were no unusual symptoms immediately, but the next morning there was elevation of temperature to 101.4 F., and a papulopustular eruption appearing over the forehead and face gradually spread over most of the body. Iodine was repeatedly demonstrated in the urine, and a diagnosis of iododerma and acute glomerulonephritis was made. Death occurred 26 days after the onset, and postmortem examination was refused. The author theorized that "although absorption from the bronchial mucosae is negligible, it is entirely conceivable that in a markedly sensitive or allergic individual a sufficient quantity could have been absorbed to be responsible for the eruption. Death, no doubt, ensued as a result of a nephritis plus an overwhelming toxemia."

### IODISM ASSOCIATED WITH OTHER DIAGNOSTIC MEASURES

*Myelography.*—A valuable adjunct in the diagnosis of lesions of the spinal cord, myelography by means of iodized oil has been criticized because of its irritating effect on the spinal meninges, because it may not demonstrate early lesions and because it occasionally shows a block where no lesion is found at operation. In their study of the immediate and late effects of the intrathecal injection of iodized oil in a series of 150 cases, Marcovich, Walker and Jessico[40] noted that there followed in slightly more than half of the cases a mild fever of short duration, less frequently headache and aggravation of previous symptoms, all of which usually subsided within 72 hours. They observed that the cells and protein content of the spinal fluid are usually increased and may remain so for several days. They used in their series 40 per cent iodine in poppyseed oil, except for a few instances in which the iodine concentration was only 20 per cent.

Numerous other authors[41-49] mentioned the principal ill effects as fever and headache. Dizziness as a rather common complaint has been mentioned.[46] Paralysis of the cranial nerves, especially of those

controlling the muscles of the eye, has been reported,[43, 45, 49] also temporary amaurosis.[50] Exaggeration of symptoms and signs previously present is not uncommon.[41, 42, 44, 46, 51-53] Occasionally there is an increase in partial paralysis.[44, 54] Marcovich, Walker and Jessico[40] were of the opinion that the severe dyspnea reported by Wartenberg,[49] and Fumarola and Enderle,[44] and also the bladder symptoms of retention and incontinence[54] as well as constipation, are exacerbations of a pre-existing condition. A case of paralytic ileus was reported by Messing.[44] In most instances these exacerbations were only transient.

In their search of the literature, Marcovich, Walker and Jessico[40] found reports of seven fatal cases. In two, there was no postmortem examination; [55, 56] of the five which came to autopsy, in two[43] there was extensive disease of the spinal cord which clouded the etiologic relationship of the iodized oil to the fatal outcome, and in the other three[46, 57, 58] acute spinal meningeal reactions were present, also in two of these similar acute inflammatory changes about the brain stem.

These authors observed that a varying number of small globules of the iodized oil are present within the intracranial cavity in approximately one third of the patients in whom the oil has been injected intrathecally. No symptoms referable to the oil, however, have been noted.[40, 59] In this connection recent animal experimentation is of interest. In 1948 and 1949, Broman and Olsson[60] demonstrated that various contrast mediums of the diodrast group, intended for use in cerebral angiography, are capable of injuring the cerebral vessels of laboratory animals by causing a disorder in the permeability of the cerebral vessels, edema, stasis and punctate hemorrhages. They observed that the incidence and degree of severity of the vascular lesions varied according to the concentration of the medium used and to the application time, namely, the time the medium was allowed to act upon the cerebral vessels. From further experimental investigations reported in 1950, Broman, Forssman and Olsson[61] concluded that repeated injection of a contrast medium into the carotid artery involves a risk of a summation-effect if the interval between the individual injections is short; that retardation of the circulation in the brain will prolong the application time and lead to an injury, even though the contrast medium is injected fairly quickly; and that latent vascular injury in the brain is liable to be aggravated and become manifest by the injection of a contrast medium of the diodrast type in cerebral angiography.

In discussing the deleterious effects of injections of lipiodol and alcohol on the tissues of the central nervous system, Verbrugghen[62]

reported four cases in which there were minor ill effects following the injection of lipiodol and added in a footnote that later he had observed a case in which severe meningeal reaction with high fever, stiff neck, coma, an exceedingly high cell count and cloudy fluid, and ultimately death followed the injection of lipiodol.

Bucy and Speigel[63] reported an unusual complication in which localized adhesive arachnoiditis at the level of the eighth thoracic vertebra, present prior to the injection of the iodized poppyseed oil, caught and held some of the oil. Stimulation of fibroblastic proliferation in the leptomeninx increased the arachnoiditis and resulted after four years in dysfunction of the spinal cord.

Tarlov[64] discussed the advantages of pantopaque over lipiodol and other contrast mediums in his report of a case of meningitis, disclosed at operation, and noted that Steinhausen and his associates[65] described the meningeal response to the injection of this medium as minimal and transient. His case, however, illustrates the danger of provoking inflammatory and proliferative meningeal reaction following the introduction of pantopaque. He advised its restricted use, its injection immediately before fluoroscopy, and its complete removal immediately after the myelography since meningeal reactions following its subarachnoid injection are more common after incomplete removal.[66] He noted also that sequelae may occur days or weeks or longer after injection and may progress as with other substances introduced into the spinal cord.[40, 63, 67, 68]

*Arteriography.*—Following the use of organic iodine compounds, severe immediate vasospastic reactions may occur in the injected limb in arteriography of the peripheral vessels. In a series of 129 arteriograms with these compounds dos Santos[69] encountered six cases in which gangrene was aggravated, but in some 300 with thorotrast he observed no vasomotor reactions. Wagner[70] described the systemic reactions as flushing of the skin with a feeling of warmth, erythematous eruptions, nausea, vomiting, cyanosis, respiratory distress and fall in blood pressure. He added that death may ensue immediately as the result of hypersensitivity or idiosyncrasy to the injected material, or else be delayed, in which instance it is thought to be due mainly to pre-existing extensive renal damage.

*Urography.*—In 1942, Pendergrass, Chamberlin, Godfrey and Burdick[71] analyzed a series of 661,800 urograms following the administration of organic iodine compounds as contrast mediums. In their survey of deaths and unfavorable sequelae, they added 26 fatalities to the 11 previously reported, an incidence of 0.0039 per cent. Ten of

the 26 were immediate and 16 delayed; seven of the 11 fatalities previously reported were immediate and four delayed. The immediate deaths followed the intravenous injection of diodrast. In the majority of these instances death was preceded by symptoms simulating those of anaphylactic shock, but may not have been due to an "anaphylactoid reaction" in the opinion of the authors. In others, they thought overdosage might have caused the fatal outcome. In the cases of delayed death, the contrast medium was not established as an etiologic factor, but they noted that toxic reactions may occur, especially in patients with previous renal damage and urea reduction. They also noted that no immediate deaths had been reported following retrograde pyelography with any of the contrast mediums. In a number of instances, nonfatal hypersensitivity, simulating anaphylactic shock was reported following the intravenous use of contrast mediums.

In commenting on toxic reactions to the contrast substances, these authors observed that they occurred chiefly in cases in which renal damage was present. They concluded from the evidence in their large series that patients with azotemia are poor risks for excretory urography, that the results of urographic studies under such circumstances are usually unsatisfactory and that there is a risk of producing a fatal reaction or further visceral damage. Stating that deaths had occurred following the use of intravenous contrast mediums in cases of nephrectomy with disease in the remaining kidney, they advised blood chemical studies prior to the use of these mediums in such cases.

They also cautioned that a suit for damages had followed death from a contrast medium in at least one instance and stated that others had been mentioned but not in detail for fear of legal sequelae. They advised physicians to heed instructions accompanying each package of contrast medium lest they be held liable for a catastrophe, even though it is well known that the use of all the preliminary tests will not warn adequately against the toxic reaction.

In the fatal case reported by Goldburgh and Baer,[72] also in 1942, death occurred within 20 minutes of the beginning of the injection of diodrast to obtain a second urogram nine days after the first one. Two outstanding features at death were the deep cyanosis and the well defined hepatomegaly, the liver being enlarged about six inches although barely palpable that morning. This fatal anaphylactic reaction was similar to the one reported by Dolan[73] in which only 3 cc of diodrast was administered intravenously. In his discussion of Crane's report[74] of two deaths attributed to the intravenous administration of diodrast, in one of which the picture at autopsy was typical of anaphylactic shock, Braasch[74] in 1939 reported 25,000 intravenous

injections of diodrast had been given at the Mayo Clinic without a fatality. In this connection, Goldburgh and Baer[72] mentioned the work of Robb and Steinberg[75] in visualizing the chambers of the heart, in which they reported almost 500 injections of from 30 to 75 cc of 75 per cent diodrast, injected within two seconds, without a serious reaction. Too, they directed attention to arterial and venous thrombosis resulting from the use of diodrast or a preparation similar to it, as reported by Boon and Lindeboom,[76] and also Cumming and Chittenden,[77] who, in addition, listed five deaths believed due to contrast mediums used in excretory urography.

### IODISM ASSOCIATED WITH IODINE THERAPY

*Iododerma.*—In the classic case of fatal iododerma reported by Eller and Fox[78] in 1931, after four months death resulted from bronchopneumonia and congestion of the liver, spleen and kidneys. This case and nine other fatalities[78, 79] were cited by Hollander and Fetterman[80] in reporting their fatal case of iododerma, which they considered the eleventh reported in the literature. There was a definite history of ingestion of iodine, usually potassium iodide, for a shorter or longer period, in all of these cases with bullous, pemphigoid or tumorous eruption developing and death ensuing from cardiac failure, bronchopneumonia or edema of the lungs in varying periods from one week to seven months. In the nonfatal case reported by Cleveland,[81] large quantities of iodine appeared in the urine after the patient was given 100 cc of a tenth-normal solution of sodium chloride intravenously. Joffe[79] thought that in serious or fatal forms of iododerma the idiosyncrasy to iodine manifests itself not only on the skin but also in the kidneys and in the central nervous system. Wolfsohn[82] was of the opinion that it is especially noted in patients suffering from exophthalmic goiter.

*Purpura.*—In 1949, Ehrich and Seifter[83] reported a case of purpura in which death resulted from the rare and uniformly fatal type of acute purpura characterized by thrombopenia and widespread formation of thrombocytic clots in the capillaries. The patient had been taking kelpidine, a commercial reducing agent containing kelp and iodine. At autopsy the yield of iodine in the kidneys, liver, lungs and heart was considerable. These authors found eight cases of purpura following iodine therapy published between 1919 and 1946.

From 1877, when Fournier[84] reported the first case of purpura following the administration of iodides, to 1904, Davis and Saunders[81] discovered only six reports in the literature. The nonfatal case they reported in 1946 resulted from administration of potassium iodide.

They regarded purpura from iodides as an ominous sign, possibly leading to a fatality if the drug is not discontinued.

*Hyperthyroidism.*—In a case of thyrotoxicosis described by Newman and Ross,[85] preoperative therapy with Lugol's solution nearly proved fatal. Severe iodism developed, the reaction appearing first in the mouth and then extending to the lungs. Also present were profuse sialorrhea and great congestion of the involved mucous membranes.

Remarking upon the infrequency of unfavorable reactions in view of the widespread use of iodine therapy in hyperthyroidism, Barker and Wood[86] described seven cases of severe febrile iodism observed in a series of 400 hyperthyroid cases in the Johns Hopkins Hospital over a ten year period, an incidence of 1.75 per cent. The one fatal case in this series illustrates the danger of continuing iodine once manifestations of idiosyncrasy have appeared. When iodism was suspected after 3 cc of compound solution of iodine had been administered for two days, the dose was doubled on the basis of reports that the symptoms of iodism may frequently be overcome by doubling the dose of iodine.[87] Because of eosinophilia and persistent fever, however, a saturated solution of potassium iodide was substituted in the hope of better toleration. The results were disastrous, and death occurred on the twenty-first day after the initial dose of iodine. At autopsy, miliary inflammatory perivascular lesions were observed throughout the majority of the tissues of the body.

Barker and Wood[86] regarded this type of fatal iodism as extremely rare. They observed that the majority of the fatalities attributed to iodine may be grouped as (1) overwhelming intoxications from the accidental or intentional ingestion of a large quantity of iodine[88] or (2) pustular or bullous ioderma occasionally developing in patients who have taken iodides over long periods.[80] The only fatal case resembling theirs which they noted in the literature was reported by Mi and Yang,[89] in which potassium iodide was given for 54 days in the treatment of a traumatic hemorrhage in the eye, with resulting high fever, erythema, abdominal pain and vomiting. The patient died in coma on the fifth day. Noting that Hoke[90] and Wolfsohn[82] reported that practically all hyperthyroid patients tested gave positive cutaneous reactions to intracutaneous injections of sodium iodide, these authors concluded that increased irritability of the skin was responsible rather than actual allergy to iodine, as Wolfsohn[82] suggested.

*Syphilis.*—Long recognized as an accident likely to occur in a small proportion of patients treated with iodine or iodides, iodism

has doubtless occurred most frequently during the course of antisyphilitic therapy. Snodgrass[91] stated that iodism is of common occurrence if the oral route of administration is used. He added that iodism was present in 12 per cent of cases when the initial dose was 5 grains (0.32 gm) or less, but in less than 1 per cent if the initial dose was more than 30 grains (2 gm).

In view of the unfavorable experiences in the various specialties with the therapeutic and diagnostic use of iodine, as revealed in a survey of the literature, the extremely severe and eventually fatal case of iodism here reported should be of interest to the otolaryngologist and also to other specialists. Iodochlorol, used in this case in the course of bronchoscopic examination, was the contrast medium employed by Storrs, McDonald and Good,[14] in their case of bronchiectasis with a granulomatous process resulting in the lung. No other specific mention of the use of iodochlorol with untoward effect was noted in an extensive review of the literature.

## REPORT OF CASE

A 62 year old white woman was admitted to the hospital on July 1, 1949 because of profound anemia and a history of hemoptysis for a period of six weeks. A roentgenogram of the chest, made prior to admission, showed an area of pneumonitis in the lower portion of the right lung.

The complete blood count was taken at the time the patient entered the hospital, and subsequent blood counts were made during the period of hospitalization. Because of the profound anemia, she received multiple blood transfusions, one on the day of admission and one on each of the two succeeding days. Following the third transfusion there developed mild urticaria, which disappeared rather promptly.

Urinalysis made three weeks prior to admission showed two plus albumin and a few red blood cells. On the day of admission, urinalysis showed four plus albumin, an occasional white blood cell and many red blood cells. Presence of albumin and red blood cells in the urine persisted throughout the illness.

Bone marrow studies led to the conclusion that the bone marrow was normal.

Roentgen examination of the skull and of the long bones gave negative results.

On July 7, under pentothal sodium anesthesia, the patient was subjected to bronchoscopy. An 8-40 bronchoscope was introduced into the trachea with some difficulty because of the short neck and the difficulty in exposing the larynx. Clotted blood was encountered at the carina, which appeared compressed from the posterior side by some extrinsic swelling. In the right main stem more clotted blood was present with extravasation of blood through the mucosa in this area, which partially blocked the opening to the lower lobe on the right side. Smears taken from this area were negative for tubercle bacilli and malignant cells. After introduction of 20 cc of iodochlorol through the bronchoscope, a bronchogram was made which gave essentially negative evidence.

There was no immediate reaction to the bronchoscopic study. The next morning, however, there developed a sudden diffuse brawny edema of the neck, definite respiratory stridor and diffuse swelling of all the mucous membranes of the mouth and tongue. The patient was experiencing acute distress. When the swelling of the neck was first noted, the submaxillary and parotid glands were

Fig. 1.—This photograph shows extensive scarring of the face with constriction of the nares. The right nasal passage was almost completely occluded. Residual scarring of the upper and lower lip is well illustrated.

especially prominent, giving the appearance of mumps or parotitis. At this time, mediastinitis was also considered, but there was neither subcutaneous emphysema nor pain in the back of the neck.

Another early symptom was hoarseness, which continued throughout the acute course of the disease. On July 10, there appeared gradually large indurated bullous pemphigoid granulomatous lesions of the face, neck, shoulders and wrists, resembling those described in reported cases of iododerma. The presence of one of these lesions beneath the tongue prevented indirect examination of the larynx because of the severe pain on any attempt to take hold of the tongue.

On July 14, the salivary glands were all diffusely swollen, sialorrhea was profuse, and ulceration of the lips and nares was extensive. The patient's condition continued to be critical. Treatment consisted of the use of antihistaminic drugs, namely, syrup of decapryn, and sodium thiosulfate administered intravenously.

Without warning, on July 18 the patient coughed up a large blood clot resembling a complete cast of the entire bronchial tree. Thereafter, profound shock of acute onset ensued. Multiple blood transfusions were given, and it was thought that tracheotomy would be necessary. There was response to conservative therapy, however, and gradual improvement in the diffuse involvement of the salivary glands and the mucous membranes of the nose and mouth followed. The cutaneous lesions, treated with Lassar's paste and silver nitrate compresses, slowly healed.

Leukocytosis accompanied the acute stage of the illness. It is noteworthy that there was evidence of renal failure at this time with elevation of the nonprotein nitrogen, which gradually returned to normal as the general condition improved. The blood count also returned to normal. With the healing of the lesions and the slow disappearance of the respiratory stridor, the excessive scarring of the lips and constriction of the nares became apparent (Fig. 1). Despite general improvement, the hoarseness persisted, owing to laryngeal scarring, as was revealed at autopsy (Fig. 2). The patient was discharged from the hospital on August 25.

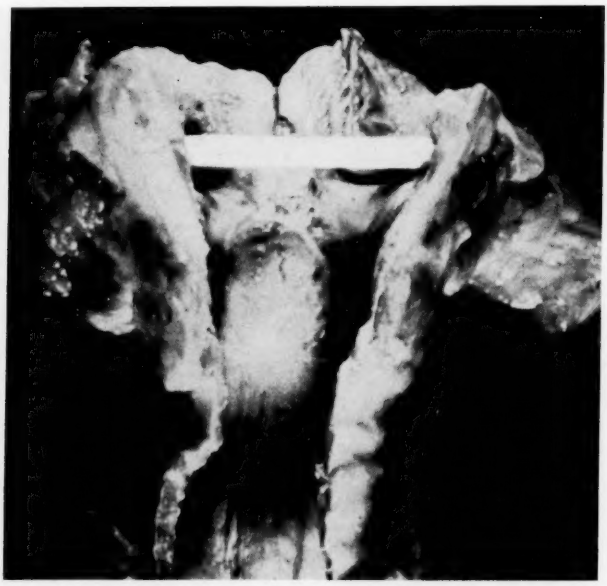

Fig. 2.—Scarring of the larynx explains the persistent hoarseness. The resultant scar would suggest great edema and involvement of the larynx during the acute stage.

She felt reasonably well for a period of one month. It was then discovered that she had again become profoundly anemic. Readmitted to the hospital on September 23, she received three blood transfusions, one on the day of admission, one the next day, and one two days later. Following each of these transfusions, hemorrhagic urticarial lesions developed on the face, especially in the areas of scar formation resulting from the previous acute illness. She was discharged from the hospital on September 29.

For a period of two weeks the patient felt well. Nausea and vomiting then became prominent symptoms. There now was evidence of azotemia, and a definite uremic odor was present. She was readmitted to the hospital for the second time on October 10. Incipient uremia was present, and headaches, generalized anasarca and convulsive seizures ensued. She died on October 29.

*Autopsy.* At autopsy, the external nares were markedly constricted, each measuring 3 mm in diameter. The bridge of the nose was also constricted and was smaller than normal. The mouth presented some contraction as evidenced by wrinkles. On the trunk and upper part of the abdomen multiple purpuric areas were scattered over-all. There were areas of diffuse scarring on the upper extremities, one on the lateral surface of the right hand measuring 10

by 3 cm. On the lower extremities multiple purpuric areas were present.

The right pleural space was completely obliterated by fibrous adhesions. The left pleural space presented multiple fibrous adhesions at the base of the lung. No free fluid was present. The transverse diameter of the pericardium measured 14 cm.

The thyroid, weighing 32 gm, was dark red and irregular. The cut surface was lobular and gelatinous.

The heart weighed 350 gm and was reddish brown, smooth and glistening on the surface. A moderate amount of yellow epicardial fat was present. The cut surface of the myocardium was uniformly reddish brown throughout. There were no abnormalities of the valves nor of the great vessels. In the aorta there were calcified and atheromatous plaques, the largest 1.5 cm in diameter.

The right lung, weighing 700 gm, was reddish grey on the outer surface and covered by dense fibrous adhesions. The cut surface was dark red and exuded dark red fluid. There was no evidence of tumors or abscess formation. The left lung weighed 530 gm and was pinkish grey and smooth on the surface with fibrous adhesions about the base of the lower lobe. The cut surface was dark red, somewhat dense and exuded red fluid. Adhesions were present on the posterior wall of the larynx just below and involving the vocal cords. The lining of the trachea and main stem bronchi was white with evidence of circular adhesions. A moderate amount of white mucus was present in the main stem bronchi, and the distal bronchi contained thick mucoid material.

No free fluid was present in the peritoneal cavity. The panniculus adiposus measured 4 cm in thickness. The abdominal viscera had their usual anatomic relationship, and no adhesions were present.

The spleen, weighing 175 gm, was bluish grey, smooth and glistening on the outer surface, and the cut surface was uniformly dark red throughout with fibrous trabeculations. There was no evidence of tumors or abscess formation.

The liver, dark greyish brown, smooth and glistening on the surface, weighed 1,400 gm. The cut surface was light brown throughout, and there was no evidence of cysts, tumors or abscesses. The gallbladder, in its usual location, contained approximately 50 cc of greenish yellow bile. No stones were present. The cystic duct was patent and communicated freely with the common duct, which in turn communicated with the duodenum.

The pancreas, weighing 70 gm, was yellow and lobular on the surface. The cut surface was lobular throughout, and there was no evidence of cysts or abscess formation.

The adrenals, weighing together 18 gm, were orange and irregular. There was no evidence of hemorrhage or tumors.

The right kidney weighed 100 gm. The capsule was adherent and stripped with difficulty, revealing a coarsely granular surface. On the cut surface, the cortex and medulla could not be differentiated, and the parenchyma measured 1 to 1.5 cm in thickness. This kidney was supplied by two renal arteries, one to the upper pole and one to the lower pole. The calyces of both kidneys were uniform and communicated freely with the pelvis, which in turn communicated freely with the ureter. The left kidney weighed 105 gm. Its capsule was also adherent, with multiple petechiae on the surface, and the appearance of the cortex and medulla was the same as that of the cortex and medulla of the right kidney. Both ureters were patent and communicated freely with the bladder, which was filled with approximately 50 cc of thick cloudy yellow urine. The mucosa of the bladder was dark reddish grey and edematous.

Present in the posterior wall of the uterus was an encapsulated solid white tumor 2 cm in diameter.

The esophagus was patent, its mucosa thrown into folds. The stomach was distended with approximately 500 cc of dark greyish brown fluid. The gastric mucosa presented the usual pattern of rugae, with no evidence of ulceration. The small intestine was patent throughout, and there was no evidence of volvulus or mesenteric infarction. The appendix, measuring 6 by 0.5 cm, presented no evidence of inflammation. The large intestine, patent throughout with the distal portion containing formed fecal matter, showed no evidence of ulceration, diverticulae, or tumors.

*Microscopic Examination.* In some areas of the lungs, there was coagulated amorphous pink staining material in the alveoli. Other alveoli contained red cells, and still others neutrophils. A sticky membrane was attached to the surface of the epithelium of the bronchi and larynx. In other areas there was fibrous replacement of the parenchyma, and in some, pigmented phagocytes.

Sections of the kidneys showed practically all of the glomeruli to be moderately to completely hyalinized. Some had thick glomerular membranes, and others were atrophic. The tubules showed desquamation of some of the lining epithelium and albuminous swelling of the tubular lining cells. In the markedly fibrosed stroma there

were numerous scattered inflammatory cells. Some of the vessels were thick-walled.

In the thyroid, no specific disease process was demonstrated.

The anatomic diagnosis was: (1) Severe chronic nephritis with atrophy of both kidneys; (2) lobular pneumonia, bilateral; (3) laryngitis and bronchitis, subacute and chronic; (4) multiple pleural adhesions, bilateral; (5) cystitis; and (6) leiomyoma of the uterus, benign.

## COMMENT

This unusual case of iodism presents practically all of the complications known to occur in iodism, except for those cases in which iodinated compounds are used in carrying out studies of the central nervous system. Glomerulonephritis was undoubtedly present prior to the instillation of iodochlorol for bronchoscopic study, and the iodism which ensued caused an exacerbation of the existing diffuse renal disease.

It is noteworthy that prior to the administration of iodochlorol, mild urticaria was manifested. Whether protein molecules were received which were sensitized to iodine is problematic. There was no history of asthma, hay fever, urticaria, or other allergy. In an unreported case previously observed by an associate, in which latent nephritis was known to exist, the administration of an iodinated compound for contrast study of the gallbladder was followed by acute renal failure, the only untoward reaction in this instance, with death resulting. It would appear that iodinated compounds may be contraindicated in the presence of latent nephritis.

In the case reported, the earliest manifestation of a reaction to the iodized solution was in the salivary glands, first the submaxillary and sublingual and later the parotid glands. Probably the most frequently observed evidence of irritation from the iodides is the appearance of eruptions on the skin, which may take the form of acne, urticaria, or occasionally pemphigus. In this case, all three were present, and from an organic standpoint the cutaneous lesions were the second manifestation. According to Webster,[92] if iodine is applied to abraded surfaces or injected into various cystic cavities, it produces general disturbances, such as headache, dizziness, delirium and collapse, and at the same time gastric irritation may occur. How the iodine was absorbed from the lung in this case is not entirely clear, but in view of the bleeding from the lung prior to bronchoscopy and the instillation of iodochlorol, it is possible that absorption took place through an abraded area within the lung itself.

SUMMARY

The important role iodine plays in the physician's armamentarium is discussed.

The therapeutic uses of iodine are mentioned, and its effect on various body tissues is reported.

The significant role of iodized oils as contrast mediums in roentgen diagnosis is presented.

Idiosyncrasies to iodinated compounds resulting in iodism are discussed. The general manifestations of iodism with respect to the skin and mucous membranes of the body are described. The deleterious effect of iodinated compounds upon the respiratory tract, the gastrointestinal tract, and the glandular organs of both internal and external secretion is discussed.

The precautions to be taken in the use of iodides and iodized oils and the contraindications to their use are outlined.

The treatment of iodism in the acute stage is discussed.

A review of the literature on iodism is presented, dealing particularly with iodism in bronchography, but also in association with other diagnostic measures, notably myelography, arteriography and urography, and, too, in association with iodine therapy.

A fatal case of severe acute iodism, with autopsy, is reported, in which iodochlorol was employed in the course of a bronchoscopic examination. It illustrates well the complications which may arise, presenting most of those known to occur, including edema of the glottis, iododerma and exacerbation of chronic glomerulonephritis.

This single case with its extreme manifestations and fatal termination serves to emphasize the risk involved in the occasional case, particularly in the presence of renal damage, as mentioned by several authors.

It is concluded that the use of iodinated compounds may be contraindicated in bronchography and other diagnostic procedures, as well as therapeutically, when pre-existing renal involvement is present.

Note: Dr. Nathaniel Jones, Internist, was associated with Dr. Irwin in the care of the patient.

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XXXV

# HEAD AND FACE PAIN OF SINUS ORIGIN

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The problem of head and face pain of sinus origin is a very complex one, both as to diagnosis and treatment. Unfortunately, it would seem that whenever the symptom of head and face pain manifests itself, the first thought is that of sinus disease. At least, this is certainly the opinion of the "man on the street" and of not a few of our own profession, as parlaying a postnasal discharge plus a headache into a diagnosis of sinus disease is still quite fashionable. Referred patients are often not a little annoyed when the otolaryngologist tells them the seat of their trouble is not the sinus, because they have been lead to believe this by the referring doctor, and this does not always lead to a satisfied patient. Contrary to popular opinion, sinusitis is not a common cause of headache and a false diagnosis of sinusitis often leads to much unnecessary treatment.

It is unfortunate that although sinus disease may not be the source of headache the close association of head pain with objective evidence of sinus disease may be present. As an example, vasomotor instability, producing changes in the mucous membrane of the nose and sinuses, may also be the cause of circulatory changes in the central and peripheral nervous systems. The sensory nerve supply of the nose and accessory sinuses is the trigeminal nerve (Fig. 1).

It will be seen from this drawing that irritation of this nerve may refer pain to any part of the head, eyes, teeth, jaw, neck or occipital region, and explains why symptoms of nasal and sinus pathology are often confused with other types of head, neck and face pain. For this reason it is difficult to separate head and face pain of sinus origin from nasal origin and, as we shall see it is practically necessary to consider both as complementary in any discussion of this subject. More specifically, the nerve supply to the nose and accessory sinuses is through the sphenopalatine ganglion area, located in the pterygopalatine fossa. This ganglion originates from three

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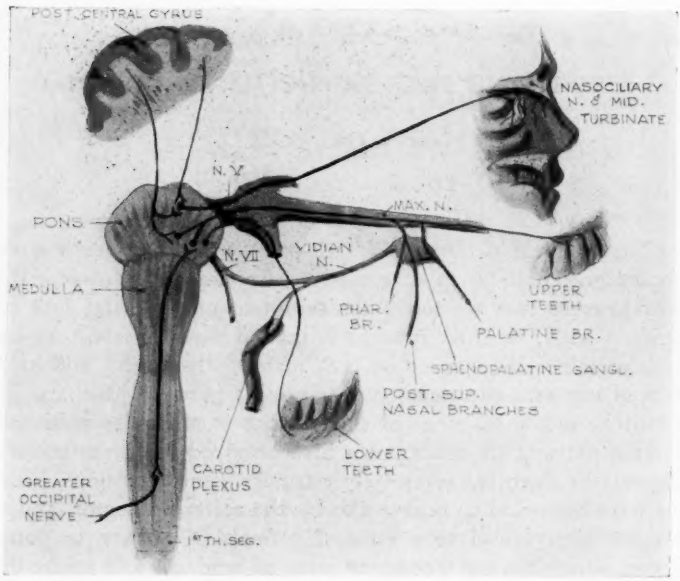


Fig. 1.—This plate shows in simple diagramatic pictures the relationship of the trigeminal nerve and synapses in the pons which make possible the complexities of referred pain.

roots: The sensory nerve from the superior maxillary division of the trigeminal nerve; the motor from the greater superficial petrosal nerve from the facial nerve; and the sympathetic great deep petrosal nerve from the internal carotid plexus. These latter two nerves run together through the pterygoid canal and form the vidian nerve on the way to the sphenopalatine ganglion.

Classically, the pain of acute sinus disease is said to be constant or periodic and sharply localized. The pain is usually of a deep, dull aching, non-pulsating quality. The headache of chronic disease is of low intensity and that of acute sinus disease is of somewhat greater intensity, but seldom very severe. The headache of sinus disease is usually reduced in intensity or abolished by aspirin or codeine. Headaches appear less frequent when the patient has been lying down for some time and are less often present at night. As a rule, headache appears in the morning and tends to regress as the day passes, although this need not be the case. Commonly, when sinus disease is of sufficient duration and intensity, there is pain in the back of the head, neck and shoulders. It is thought that this

referred pain never results from direct stimulation of the nose or sinuses, but that such pain is due to secondary effects of prolonged contraction of cervical or scalp muscles. It has been shown experimentally that pain in the back of the neck is a common symptom from painful stimulation of any part of the head over a long period.

Pain due to sinus disease is aggravated by stooping, exertion, sudden movement of the head, sneezing or coughing. This is probably due to increased venous pressure, which accompanies such exertions. The pain is also intensified by states that increase the engorgement of the mucosa, such as anxiety, resentment, cold air, menstruation and alcohol.

Acute maxillary sinusitis produces local discomfort and referred pain to the cheek, upper jaw, upper teeth and frontal area. There is local tenderness of the upper teeth on chewing. It is said the pain of maxillary sinus disease gradually diminishes as the patient assumes a reclining position with the disease side uppermost.

Headache is the most common symptom of acute frontal sinusitis. The pain is situated directly over the sinus in the supra-orbital region, the eye, or root of the nose, and may even encompass one-half of the head. It may produce nausea and it is often throbbing in character.

Anterior ethmoid involvement produces pain at the root of the nose and inner angle of the eye, and the pain from the posterior cells may be referred to the temporal region, front teeth and occasionally to the parietal and occipital regions.

In any discussion of pain in this area I feel we must also recognize the pain of a sharp septal spur digging into an intumescent middle turbinate. This condition also produces pain at the root of the nose, inner angle of the eye, and may even be referred to the front five teeth.

Acute sphenoiditis produces deep pain behind the eyes, deep in the head and over the vertex, and occasionally into the mastoid area.

Headaches from chronic sinus disease are much less easy to define. Usually they are more general, less constant and only assume the typical picture outlined above during exacerbations.

I feel it would not be amiss to briefly review the findings from the work of McAuliffe, Goodell and Wolff,[1] which was presented in the proceedings of the Association for Research in Nervous and Mental Disease, in 1942.

Stimulation of the nasal septum produced, besides local pain, discomfort in the region of the zygomatic eminence, the inner canthus and upper teeth back to the ear on the same side. Stimulation of the lateral wall of the maxillary sinus produced pain in all these same areas, except the inner canthus.

Stimulation of the inferior turbinate produced local pain, plus referred pain to the upper teeth and zygomatic region of the same side. Irritation of the middle turbinate produced pain in the upper teeth and zygomatic region and stimulation of the superior turbinate produced pain over the lateral wall of the nose, inner canthus and glabella.

Stimulation within the sphenoid sinus caused referred pain to the vertex. Strangely enough, no pain was referred to the occiput, as is generally accepted for disease of this sinus.

Stimulation of the superior nasal wall in the region of the anterior ethmoid cells referred pain to the eye, outer brow and just lateral to the ala on the same side. Stimulation of the posterior ethmoid region produced pain at the outer brow, under the outer canthus, and from the ala over the upper alveolar ridge. This pain was accompanied by lacrimation and photophobia. Stimulation of the region of the sphenoid produced only local pain.

Stimulation of the nasofrontal duct produced pain of severe intensity over the inner canthus, maxilla and zygoma, extending into the temporal region. Pain was also referred to the last two upper molars. Irritation of the frontal sinus produced only local pain over the sinus, of less intensity. However, profuse lacrimation and conjunctival injection accompanied this stimulation.

Stimulation of the maxillary sinus ostium produced pain over the zygoma, temporal region, the last three upper teeth, and the tonsillar area of the pharynx on the same side. This was also accompanied by profuse lacrimation and conjunctival injection.

The maxillary sinus was examined through an oral-antral fistula, and pressure applied internally to the maxillary sinus walls produced pain in the upper teeth, maxilla, eye and supra-orbital region.

Now I think we come to the real significance of the stimulations outlined: It is interesting to note that pain from direct stimulation of the sinuses was of low intensity, while pain from stimulation of nasal structures was three to four times greater, even in corresponding referred areas.

Further experiments were carried out by placing a balloon into the antrum through the oral-antral fistula. It was necessary for the pressure to reach 200 mg of mercury before pain was felt in the sinus. It was noted that maintenance of this pressure in the sinus produced engorgement of the turbinates and after they reached the stage of occluding the nose only 50 mg mercury pressure was necessary to produce comparable pain. This pain could be stopped, or greatly reduced, by applying topical anesthetic to the turbinates. It may be inferred, it would seem to me, that sustained intra-sinus pressure produces little pain, but does produce reflex engorgement of the turbinates, and it is this condition that produces the pain. Creating a vacuum produced no pain until 250 mg mercury negative pressure was obtained. This was thought to be due to the stimulation of the ostium, as removal of the cotton tamponade showed it to be drawn into the ostium. One can postulate that negative pressure in a sinus, whose ostium is blocked by thickened membrane, could produce pain, not from the vacuum per se, but from irritation of its ostium.

It will be seen that the membrane of the sinuses is relatively insensitive as compared with the ducts, ostia and turbinates. Anesthesia of various nasal structures tested reduced the referred pain and several patients with sectioned trigeminal nerve were examined and they had no pain whatsoever. It was noted that subjects with engorged turbinates and nasal mucosa, when stimulated, complained of more intense pain, and over a larger referred area, than those with normal looking mucous membrane in the nose.

I feel that we must accept the fact that nasal obstruction causing pressure on nasal structures will cause pain and is much more important than the changing pressure within the sinus. This might explain why patients using vasoconstricting nose drops feel relief even though we know they have done little to actually drain a sinus.

## SUMMARY

I have attempted to outline a modern concept of head and face pain of sinus origin, without entering into a discussion of pathology, clinical findings or treatment. In spite of the classical pictures outlined, one must remember the tremendous individual variation in the production of pain. No doubt, variations in anatomy play a part, but we know that a sinus full of pus, even without drainage, will cause little or no pain in one person and extreme pain in another. Also, we see a patient with a nose completely blocked with polypoid tissue suffering no real pain, and another patient with mild intumescent obstruction complaining bitterly. I feel that these differences

probably lie in the complexity of synapses in the sensory network to the nose, accessory sinuses and head, and to the degree of alteration of the normal physiology of the erectile tissue in the nose.

The interpretation of head and face pain attributable to sinus disease has probably produced more headaches for the doctor than sinus disease has for the patient.

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XXXVI

# HEAD AND FACE PAIN DUE TO AUTONOMIC DYSFUNCTION

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The role of the autonomic nervous system in the production of head and face pain has justifiably been given greater prominence in recent years. This is understandable in view of the shift in emphasis in medicine from the older anatomic to the newer physiologic approach to disease processes and symptoms. Thanks to this approach the individual is now looked upon as a vibrant, changing mechanism constantly adjusting to an ever varying physical and psychic environment, instead of a collection of semi-isolated normal or pathological organs functioning in an independent and mechanical manner.

Among the concepts gaining importance in this newer view in medicine is that of the autonomic nervous system—the regulator of the body's response to change. It is the correlator of the body's attempts to maintain homeostasis or the status quo. This portion of man's nervous system has also been called the vegetative nervous system controlling as it does the involuntary or automatic responses of the body. Since the maintenance of homeostasis requires a delicate balance in physiological processes there is necessary a system of check and counter-check in these processes. The countering processes receive their innervation through the opposing subdivisions of the autonomic nervous system—the sympathetic or adrenergic and the parasympathetic or cholinergic divisions.

Major functions controlled by the autonomic nervous system in the head and face are those concerned with circulation and glandular secretion. It is with the circulation that we are concerned in our discussion of pain.

Vasoconstriction is mediated by sympathetic stimulation and vasodilatation by parasympathetic stimulation. Both these phases of vascular response to autonomic stimulation can produce pain.

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The findings of Odermatt,[1] Spiegal and Wassermann[2] indicate that distention of blood vessels gives rise to pain due to the effect of distention on the periarterial nerve plexus. Conversely, strong peripheral vaso-constriction not infrequently is accompanied by pain. Evidence is not wanting which seems to support the theory that this pain is caused by the contraction of the vascular musculature. Other evidence seems to indicate that the ischemia produced in the tissues by the contraction of the blood vessels may be a contributing factor in the causation of pain. Other experimental and clinical data seems to support the assumption that accumulated metabolites in ischemic tissues may stimulate the pain receptors closely associated with the smaller blood vessels. It would seem, however, that, in general, vasodilation is the more important process in the production of vascular pain than is the vasoconstrictive phase.

Vasospasm followed by vasodilatation come into play though as the fundamental process in all vascular headaches and pains, whether they be called migraine, histaminic cephalgia, carotid or vasodilating pain, allergic headache, or cervical myalgia. The vasospasm occurs in some vessel (usually at arteriolar level) as an autonomic response to some poorly handled internal or external stimulus. There follows local ischemia to the part beyond; which, if the process be reduced to its simplest and smallest unit—arteriole-capillary-vennular unit, causes damage to the endothelium of the capillary with subsequent dilatation and fluid leak into surrounding tissue spaces.

Depending upon the site of the arterioles involved the effect upon the parent artery varies. If the spastic arterioles are terminal they erect an effective dam which causes vasodilatation proximal to them. Or if the arteriolar vasospasm with subsequent dilatation and capillary leakage occurs in the vaso-vasorum of the subject artery wall as Hilger[3] pictures it, then one can see the subsequent edematous dilatation of the parent artery's wall. Either situation—the pulsating artery swelling as blood, pounding through it, comes up against the impasse of the constricted arteriole beyond or the artery dilating because of edema forming in its walls as a result of vaso-vasorum spasm —stretches the periarterial sensory nerve plexus and is experienced as pain whose locale depends on the vessel involved and the cortical pattern of pain representation associated with whatever vessel it be. Harold G. Wolff,[4] in his excellent investigation of the relative pain sensitivity of the structures of the head, concluded that of the tissues covering the cranium, all were more or less sensitive to pain, the arteries being especially so. Of the intracranial structures, the great venous sinuses and their tributaries from the surface of the brain,

parts of the dura at the base, the dural arteries and the cerebral arteries at the base of the brain, the fifth, ninth and tenth cranial nerves, and the upper three cervical nerves were sensitive to pain. Those not sensitive to pain were cranium (including the diploic and emissary veins), the parenchyma of the brain, most of the dura, most of the pia-arachnoid, and the ependymal lining of the ventricles and the choroid plexuses. He also concluded that pain or headache arising from dilatation of the cerebral arteries above the tentorium cerebelli had as its principal afferent pathway the fifth cranial nerve and was felt in the frontotemperoparietal region of the head; whereas pain originating from dilatation of the cerebral arteries of the posterior fossa had as its most important afferent pathways the ninth and tenth cranial and the upper cervical nerves and was felt in the occipital region of the head. Pain arising from dilatation of extracranial vessels tends to be located more in reference to its source than does pain of similar intracranial origin, although McAuliffe, Goddell and H. G. Wolff[5] have demonstrated reference of pain of nasal and sinus origin to surrounding extracranial areas.

One must remember that any of the many vessels or branches of vessels in the head and neck may be involved in vascular pain so that the clinical picture may be quite variable. The basic physiopathology is the same though whether the offending stimulus be emotional tension climatic variables such as dampness, drafts or changes in the weather, some inhalant or ingestant allergies, or merely fatigue.

A brief description of the classical types of head and face pain which fall into the category of vascular pain follows.

Perhaps the most widely known type of vascular or autonomic headache is migraine. Migraine classically refers to a relatively severe headache which usually, but not necessarily, lasts for several days. It is reputed to have a familial tendency. It occurs more frequently in women than in men and generally appears between puberty and the menopause, but is not apt to occur in the latter half of pregnancy. Typically it is unilateral and is usually preceded by an aura. This may be ocular or aural and not infreqently numbness or paraesthesias occur in the extremities transiently before the onset of the headache. The onset of the individual headache is usually gradual and the pain tends to be unilateral and pulsating. It may occur anywhere in the head but most commonly the temporal area is involved. Later the pain spreads to both sides of the head. The victim's mood is usually one of depression during the attack and nausea and vomiting may occur at its height.

It has been shown that the intensity of the migrainous pain varies with the amplitude of pulsation of the cranial arteries, chiefly those of external carotid origin.[4] Efforts to reduce the pulsation through the vessels gives relief. Thus manual compression of the carotid artery or pharmacological constriction of the lumen of the vessel by agents such as ergotamine are helpful.

It is felt that the external carotid subdivisions—numerically superior to those of the internal carotid both in the meninges and subcutaneously—are primarily involved in migraine because raising the intracranial pressure experimentally does not relieve the pain as should be the case if the pial and cerebral arteries were the major contributors to the pain. However, that the branches of the internal carotid are also involved is suggested by Wolff,[4] in that he finds that faradic stimulation of the proximal few centimeters of the anterior, middle and posterior cerebral arteries and the first few centimeters of the intracranial portion of the internal carotid artery causes pain within, behind, and over the homolateral eye. Moreover, he finds that stimulation of the vertebral and basilar arteries and the proximal portions of their branches causes pain in the occipital and suboccipital regions—areas commonly painful during the later stages of migrainous attacks. Furthermore, since the areas supplied by the superficial frontal, supraorbital, and anterior meningeal arteries, which are branches of the internal carotid artery, are commonly involved in migraine, it seems presumptive to limit the involvement in this condition to the branches of the external carotid artery alone. Another indication that cerebral (internal carotid) blood flow is involved in migraine is Wolff's[4] finding that scotomata disappear upon administration of the cerebral vasodilator amyl nitrate to the individual.

Histamine cephalgia was first reported and its differentiation from migraine was stressed by Horton and his associates in 1939.[6] Typically it is said to be a severe boring pain of relatively short duration (usually less than an hour) waking the victim in the middle of the night. The pain is unilateral involving the region of the eye, temple, neck, and often the face. The pain is eased by sitting up or standing and does not usually induce nausea or vomiting. Increased surface temperature, lacrimation, conjunctival injection, nasal stuffiness, and a watery rhinorrhea on the side involved are supposed to be characteristic. The condition occurs in men more frequently than in women and is more apt to occur in middle and later life. The intracranial vessels are felt to be primarily involved.

This type of headache is supposed to be due to a release of histamine or a sensitivity to the drug, but the mechanism of such action is obscure. Affected individuals react to subcutaneous injection of .1 to 1.2 mg of histamine base by development of a typical headache within 30 minutes. This, of course, follows the usual physiologic headache and the pain free interval which follows the headache which ordinarily occurs following histamine injection regardless of histamine sensitivity. Therapy has included both subcutaneous and intravenous "desensitization" to histamine. Horton[7] and others[8] clearly describe dosage schedules.

Whereas typical histaminic cephalgia is quite rare, "carotid pain" as described by Hilger[9] is commonplace. This term "carotid pain" is somewhat loosely applied to facial, head and neck pains expressing themselves along the distribution of the external carotid artery. Again the discomfort arises from vasodilation and may be present in a swollen painful carotid trunk itself (often erroneously diagnosed as swollen glands) or may be more peripherally manifest in any of the branches of the external carotid artery. Thus one commonly sees vasodilating pain in the region of the superficial temporal artery, the posterior auricular or occipital arteries, or the internal maxillary artery or its more commonly involved branches—the infaorbital, the posterior superior alveolar, and the sphenopalatine arteries. Vasospasm arising in these various vessels accounts for much of the pain in the facial, head and neck regions for which no other more definite pathology such as sinusitis, dental caries, or otitis media can be found. Thus we have ear, dental or sinus pain without demonstrable pathology. If the involved vessel is superficial—as the external carotid or superficial temporal arteries—it may be tender and swollen. If the vessel is more deeply protected by overlying structures, as are the branches of the internal maxillary artery, then the swelling and tenderness of the vessel can only be surmised. However, in most of these cases compression of the external carotid artery (i.e. blocking the thrust of blood into the dilated artery beyond) will give the patient some comfort. It is probable that most of the sphenopalatine, Vidian and other neuralgias of the past fall into this category.

Allergic headache, which is generally regarded as a rarity, represents autonomic dysfunction at the basic arteriole-capillary-venule level from antigen-antibody reaction with vasospasm, tissue damage, and histamine release as the usual concept.

Myalgia of the cervical musculature would seem to be frequently due to autonomic dysfunction. There are, of course, other conditions

causing muscle discomfort in these areas such as strain and fibrositis, but the occurrence of tight tender muscles in the cervical area in individuals with other vasomotor problems (vasodilating pains, vasomotor rhinitis, hydrops of the labyrinthe) suggests autonomic imbalance as the source of many of these neck discomforts.

Exposure to drafts, dampness, changes in the weather, or air conditioning are apt to produce painful stiff neck muscles in individuals with physical sensitivity or physical allergy according to Williams.[10] He lists the trapezius, splenius capitis, sternocleidomastoid, stylohyoid, superior constrictor of the pharynx, glossopalatinus, and the muscles attached to the alae of the thyroid cartilage as being commonly involved. Obviously, a variety of neck and pharyngeal symptoms can result. An example is the sore throat with no sign of infection. That the basic problem is one of vasospasm in the arteries or arterioles in the musculature involved seems apparent from the relief obtained by the use of vasodilators such as nicotinic acid and histamine. That painful, stiff, neck musculature produces headache by pulling on the occipital and frontalis musculature connected through the galea aponeurotica was stressed years ago by the German otolaryngologists.

Therapy for all of these conditions, whether it be migraine, histaminic cephalgia, carotid pain, allergic headache or cervical myalgia, must be directed toward stabililizing the autonomic nervous system and the disturbed vascular system innervated by it. Important, too, is repair of the ischemically damaged endothelium. Stabilization of the autonomic nervous system may be established by blockade of that system at central, ganglionic or myoneural levels.[3] Central blockade is accomplished by sedatives (barbiturates) and analgesics (opiates, aspirin, etc.). Ganglionic blockade may be produced by procaine or tetra-ethyl-ammonium chloride. The myoneural junction may be blocked by the antihistaminics, artane, intravenous procaine, bentyl, or the ergotamine derivatives. Direct action on the smooth muscle surrounding the offending arteriole is also important and may be accomplished with nicotinic acid, roniacol tartrate, papaverine, paveril, the nitritoid group and histamine. Endothelial repair agents include the steroids, thyroid and ascorbic acid. Perhaps balancing the steroids may find a major place in therapy when one considers the association of many headache symptoms with phases of the menstrual cycle plus the fluid-retaining and edema-producing properties of the hormonal steroids.

All of these drugs have their place and should be used as experience with the individual patient directs. The most practical for

long term prophylactic daily usage in most of these conditions seems to be oral nicotinic acid given in mildly flushing doses about four times daily. In the migrainous individual ergotamine products such as gynergen and cafergot have a popular place in preventing vasodilatation if given during the vasospastic (scotomatous or paraesthetic) stages. Octin is also used in this regard.

By its very nature all drug therapy has certain limitations. Basically the answer in many vasomotor head and facial pain problems is the adjustment of the individual to his environment. The tensions and conflicts inherent in modern living often need resolution in these individuals; and, accordingly, the psychosomatic approach is fundamental.

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XXXVII

# GLOMUS JUGULARE TUMOR OF MIDDLE EAR WITH NORMAL DRUM; IMPROVED BIOPSY TECHNIQUE

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Guild[1] in 1941 described a primary neoplasm invading the middle ear arising either from the adventitia of the jugular bulb below or along Jacobson's nerve on the inner tympanic wall. This slow-growing tumor may remain localized in the tympanum for a long time but as a rule, when it is first recognized or suspected, it has already perforated the drum and invaded the mastoid bone, petrosa, and intracranial contents. In the external auditory canal the tumor appears as a bleeding polypoid mass surrounded by foul, purulent discharge as if the entire process were a virulent type of chronic purulent otitis media and mastoiditis. Microscopic examination reveals the neoplasm. The clinical picture, which may exist insidiously for several years, often presents the following: chronic otorrhea, bleeding polypoid mass in the external canal, pain, tinnitus, vertigo, progressive loss of hearing, and even cranial nerve involvement as the tumor spreads. Successful surgical excision is possible before the tumor spreads.

About 60 cases in all have been reported in the literature. Very few of these have been suspected or reported before the tumor had perforated through the drum and spread to neighboring structures. Jeschek[2] observed a glomus tumor for eight years before it appeared in the external auditory canal. Goekoop[3] reported similar tumors in three sisters in two of whom the drum was intact. Lundgren[4] reported three cases with roaring tinnitus, slight deafness and bulging drum. Lattes and Waltner[5] reported two cases with intact but bulging drum. In one of these the drum was split for biopsy removal amid profuse hemorrhage. In both of their cases surgery revealed extension into the temporal bone. Bartel's[6] cases have not been available for review. Brown[7] reported a glomus tumor arising from the promontory with intact but bulging drum.

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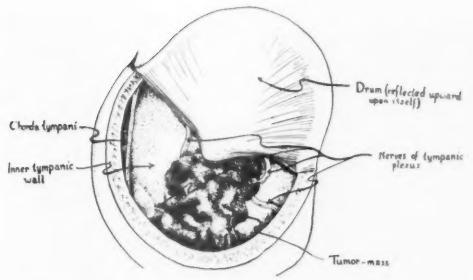


Fig. 1.—Exposure of tympanic tumor to view. The drum is lifted out of its sulcus and reflected upward.

The purpose of this paper is to report three additional cases of glomus tumor of the middle ear with intact drum (two cases with a normal flat drum and one case with a slightly bulging drum), and an improved technique for biopsy employed in these three cases. The first case, in which the drum was flat and normal, was seen in New York and will be described in detail. The second case with a flat, normal drum and the third case with an intact but slightly bulging drum were seen in Israel while on a medical mission for the Israeli Government in July, 1951. The latter two cases will be reported by the Israeli otologists. Microscopic studies revealed a glomus jugulare tumor in all three cases.

## CASE REPORT

L. T., a 39 year old female was first seen April 12, 1951, complaining of constant, severe, low-pitched roaring tinnitus together with a high swishing tinnitus in the left ear for two years. During this period there was spasmodic, recurrent tic-like pain deep in the ear with left hemicrania. The hearing was at 15-20 decibels for air and bone conduction. Physical, neurological and laboratory examinations were negative. Taste was normal. Otoscopic examination of the left ear revealed a normal canal and a normal flat drum. Through the drum a reddish-purple, sharply circumscribed mass could be seen in the middle of the tympanic cavity about 5 mm in diameter, extending upward from the region of the tympanic floor to just above the umbo.

For biopsy,[1] an incision is made through the skin of the bony canal wall from "three to nine o'clock" about 5-7 mm external to the drum. The incised skin is separated from the bone as far as the drum. The drum is then lifted out of its sulcus and reflected up-

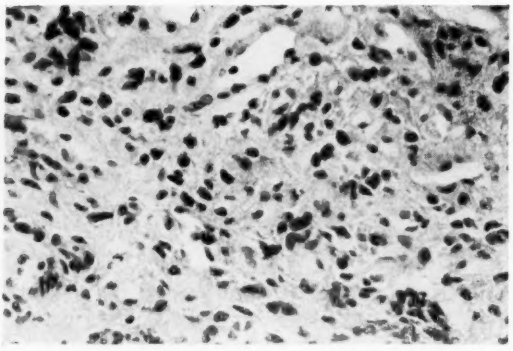

Fig. 2.—Photomicrograph; glomus jugulare tumor; high power magnification.

ward upon itself, thus exposing the lower half of the tympanic cavity. This technique has been used by the author for section of the chorda tympani nerve for the Ménière's disease.[8] In general this technique is similar to that used by Lempert[9] for the relief of tinnitus aurium.

On April 20, 1951, the above technique was employed. There was a soft, firm, purple, vascular, discrete and circumscribed mass which extended upward from the floor of the tympanum to above the umbo. It was 5 mm in diameter and did not involve the inner wall of the ear drum. A specimen was removed with punch forceps. The bleeding was easily and quickly controlled. The drum and attached skin were then placed back in their normal positions (Fig. 1).

The architectural pattern of the mass resembles a fibromatous structure. The cells have an extremely obscure cell boundary and the nuclei lie in a very fine fibrillar structure somewhat resembling glial elements. The tumor is very well vascularized by thin-walled capillaries and has an occasional thick-walled arteriole. The nuclei are mainly of spindle shape with rounded ends. They vary considerably in size and in some nests they are very large, hyperchromatic, and show considerable anisocytosis. Mitoses are not seen. The cytoplasm tends to be pale and frequently has minute vacuoles. In some portions, where the cell membrane is fairly distinct, the cytoplasm is quite clear. The surface epithelium is preserved, and is low columnar in type. Diagnosis: non-chromaffin paraganglioma of tympanum (Fig. 2). These sections were seen and the diagnosis confirmed by Drs. Lisa, Klemperer, Otani and Eggston.

On April 24, 1951, a radical mastoidectomy was performed. The tumor arose from the promontory and had not spread to the temporal bone. The bony floor of the tympanum was intact. The tumor was removed without any difficulty and with little bleeding.

SUMMARY

Three cases of glomus jugulare tumor of the tympanum with intact drum are reported. In two of the three cases the drum was flat and normal. The technique described for biopsy exposes the tympanum for inspection of the extent of the tumor mass and avoids the severe hemorrhage occasioned by incision of the drum for biopsy. In cases in which the tumor arises from the promontory it might be possible to excise the entire tumor by the technique described and thus avoid radical mastoidectomy.

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XXXVIII

# HEMATOMA OF THE LARYNX FROM EXTERNAL TRAUMA

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Permanent damage to the larynx from external force is infrequent. Unless powerful force is exerted directly upon the larynx from without it usually escapes injury because of its mobility over the cervical vertebrae. Protection is also offered to the larynx by the mandible, and the lack of ossification of the cartilaginous framework tempers any trauma to the organ. Contusion or hemorrhage may exist without detection. If there has been injury to other parts of the body primary attention is focused elsewhere rather than upon the neck and larynx. Contusion, hemorrhage and even fracture of the laryngeal cartilages occur after trauma, and it is difficult to know how much damage has been done to the larynx in most injuries, for unless the symptoms are directed solely to the neck or larynx inspection of the interior of the larynx is usually overlooked.

Aside from fractures of the thyroid or cricoid cartilages and gunshot and penetrating wounds of the larynx, submucosal hemorrhage and hematoma formation may develop from external blunt force. Although hematomas form from vocal abuse, infection and endotracheal intubation these lesions are not related to external trauma. Cox and Van Eycken[1] reported a case of traumatic hematoma of the larynx as a result of a football injury. A hematoma of the larynx due to an explosion of a gas stove with indirect injury to the larynx from a flying piece of metal was reported by Lumsden.[2] Jackson and Jackson[3] commented on the dearth of literature regarding laryngeal trauma and listed 19 cases resulting from blows on the neck or larynx. Six additional cases are included in this report (Table 1).

Even though traumatic hematoma of the larynx from external force is infrequent, I do not believe a true picture of the incidence has been recorded, for most laryngologists have encountered this condition at sometime but have not reported their findings. Blows

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Read at the combined meeting of the Sections of Otolaryngology of the Academy of Medicine of New York and the College of Physicians of Philadelphia, Philadelphia, Pa., March 19, 1952.

TABLE 1.—CASES OF LARYNGEAL HEMATOMA PRODUCED BY EXTERNAL BLUNT FORCE.

| AGE & SEX | METHOD OF INFLICTION | SYMPTOMS | PORTION OF LARYNX INVOLVED | MOTILITY | RESULT |
|---|---|---|---|---|---|
| M/39 | Blow on neck in fight | Neck tender<br>Dysphagia<br>Blood spitting | Rt. V. C., arytenoid lat. pharyngeal wall | Impaired | Imp. 1 wk. |
| M/24 | Iron bar fell across neck | Dyspnoea<br>Odynphagia<br>Fullness front of neck | Lt. V. C., vent. band, prolapse of ventricle, base of epiglottis | Normal | Imp. 1 wk. |
| M/15 | Blow on neck in football | Odynphagia<br>Pain in throat | Rt. memb. V. C. | Normal | Well 2 wks. |
| M/28 | Automobile accident | Odynphagia | Both V. C., rt. arytenoid, aryepiglottic fold | Normal | Well 1 mo. |
| M/63 | Plank fell on neck | Hoarseness<br>Dysphagia<br>Neck contusion | Lt. vent. band, arytenoid, aryepiglottic fold | Impaired | Imp. 2 wks. |
| M/45 | Blow on neck in fight | Hoarseness<br>Odynphagia | Both V. C., lt. aryepiglottic fold, vent. band | Impaired | Imp. 2 wks. |

upon the larynx from bodily contact, either from a fist or knee, in fighting or athletic contests, are the most frequent cause of indirect laryngeal damage (Fig. 1). Falling objects and accidental falls constitute other sources of laryngeal damage.

Painful swallowing is a prominent symptom and causes the patient considerable discomfort. The act of swallowing with or without food and fluid produces pain in the laryngeal area, sometimes referred to the ear. The pain occurs during deglutition, and the patient is free from pain during the intervals between swallowing.

Varying degrees of hoarseness are frequently present, but unless there is pain on speaking this symptom causes the patient little concern. In extensive hematoma, aphonia or a marked degree of hoarseness accompany painful swallowing. Because of swelling, difficulty in breathing may be present, but rarely is the obstruction marked enough to necessitate tracheotomy. Occlusion of the airway if it occurs commonly develops rapidly, so that when tracheotomy is needed after hemorrhage prompt operation is impertative. Rarely

Fig. 1.—Mirror appearance of hematoma of the right vocal cord resulting from a blow on the neck in a football game.

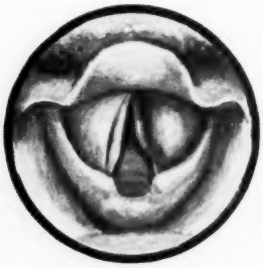

Fig. 2.—Appearance of hematoma of larynx by mirror laryngoscopy produced by a large iron bar falling across the front of the neck. The left side of the larynx exhibits a large submucosal hemorrhage, involving the left vocal cord and ventricular band with prolapse of the mucosa of the ventricle.

Fig. 3.—Mirror laryngoscopic appearance of an extensive laryngeal hematoma involving both vocal cords, left ventricular band, arytenoid, aryepiglottic fold and epiglottis with extension into the pharynx, resulting from a blow on the neck by an unknown assailant. Fixation of the left side of the larynx persisted for 5 days.

does the bleeding ensue slowly and gradually block the airway, but rather has a sudden onset and after the hematoma once forms it tends to remain self limited and does not increase in size. This is in contrast to the laryngeal bleeding which occurs in purpura and hemophilia in which tracheotomy is nearly always needed to provide an adequate airway, and the hematoma progressively enlarges.

Unless the force applied to the larynx is sufficient to cause laceration of the mucosa of the interior of the larynx spitting of blood is absent. A history of shock or loss of consciousness after the injury may be obtained.

Swelling over the external larynx or neck occurs at times. Palpation of the larynx and especially lateral movement over the vertebral bodies is extremely painful with accompanying tenderness on slight pressure over the thyroid cartilage. Discoloration of the soft tissues of the neck may or may not be observed. Subcutaneous emphysema and crepitation are absent unless the mucosa has been lacerated, and this commonly is associated with fracture of the thyroid and cricoid cartilages.

Mirror examination of the larynx reveals a dark red or purplish color in that portion of the larynx involved by the hematoma, which may include the epiglottis, aryepiglottic folds, valleculae, ventricular bands, vocal cords, arytenoids and extend into the soft tissues of the pharynx. Swelling is present, but is most prominent when the vocal cords and ventricular bands are involved (Fig. 2). These changes may be bilateral but more often are unilateral, for the productive force usually hits one side of the triangular thyroid projection rather than the center. The hematoma may be localized with sharply demarcated edges, but generally the intense red or purple discoloration fades toward the periphery gradually approaching the normal mucosa (Fig. 3).

Fixation of the involved side, when it occurs, results from local tissue damage in the musculature rather than actual involvement of branches of the recurrent laryngeal nerve. In these cases there was fixation in three cases and unimpaired motility in the other three. Movement on the fixed side returned in 24 hours in one case with complete restoration of motion in several days. One would expect a much longer period of fixation if the primary damage was to the nerve trunk or its endings. No permanent laryngeal damage followed the return of function in the cases with fixation.

Roentgen examination offers little aid in the diagnosis. The presence of fractures of the thyroid or cricoid cartilages may be elim-

inated, but are not discernable on the roentgenogram in many instances.

Since the airway is rarely impeded in uncomplicated traumatic injury sufficiently to require tracheotomy the primary treatment is vocal rest and avoidance of any form of irritation. After the larynx has been put at rest, absorption of the hematoma occurs, so that in 2 weeks time resolution often is complete with no permanent disability resulting. Attempts at aspiration are meddlesome and carry the risk of introducing infection. Antibiotics are not indicated unless the membrane has been lacerated, and then are used to prevent or combat infection. To try to accomplish sterility by medication when the blood itself is already sterile is futile. Since further hemorrhage into the tissues of the larynx does not develop after the initial injury blood coagulants are not used.

The pain may prevent swallowing and intake of adequate nourishment unless analgesics and sedatives are employed. Passage of a feeding tube is usually not necessary, and local medication has proved of little value except to reduce pain. The odynphagia rarely lasts longer than 72 hours, and generally food intake is curtailed only for that length of time.

## SUMMARY

Six cases of traumatic hematoma of the larynx produced by external blunt force are reported. Odynphagia and hoarseness were the principal symptoms. The hematomas varied in extent from localization to one vocal cord to involvement of the entire larynx with extension into the pharynx. No permanent laryngeal damage resulted even though in 3 cases the trauma was severe enough to cause fixation of one side of the larynx.

My appreciation is expressed to Dr. Robert M. Lukens for the illustrations.

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# XXXIX

# REMOVAL OF THE PETROUS BONE FROM THE CRANIAL BASE FOR MACRO- AND MICROSCOPIC INVESTIGATION

## HISTORICAL AND TECHNICAL CONSIDERATIONS

GEORGE KELEMEN, M.D.

BOSTON, MASS.

At the meeting of the Pathological Society of London, February 15, 1853, Joseph Toynbee lectured on "The mode of dissecting the ear for the purpose of pathological investigation." In the hundred years since this presentation much thought has been given to the subject of his lecture with accumulation of considerable literature.

It could not be otherwise. The first step in the long and laborious process of preparing the organ of hearing for histological examination is the removal of the temporal bone, or merely the petrous pyramid from the cadaver. Without a planned method and careful execution thereof, the consequences of any damage done at the removal will show up under the microscope in the finished section, six months or more later. It is only natural that much attention has been given to the technique of this first and decisive procedure.

The following are a few excerpts from the pertinent literature on the technique of temporal bone excision:

Toynbee (1853) removed the calvarium, then sawed the temporal bone free on both sides from without, inward. The saw cuts extended inward to the body of the sphenoid and the basilar portion of the occipital bone. Then the apex of the petrous bone was separated from the sphenoid and occipital bones with the chisel or saw. The external ear and integument were detached, drawn upward with as much of the soft tissue as possible.

Klebs (1869) quoted Lucae who advocated a start with a narrow chisel and the placement of two downward blows, one be-

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tween the medial and anterior clinoid processes, and the other through the center of the clivus of Blumenbach. A small saw was then used to encircle and free the temporal bone.

Wendt (1872) made a deep furrow with a broad chisel along a line connecting both lesser wings of the sphenoid, and then completed the removal with a narrow, round-pointed saw. A limited use of the knife was necessary, but only at the base. Wendt claimed that with some experience this could be accomplished in five minutes without disfigurement of the head. He further claimed satisfactory results in more than 1100 cases within a period of three years. Moreover, Wendt was the first to ascertain the requirements for histologic examination. His early death cut short his investigations.

The three authors mentioned above gave minutely detailed advice on the meticulous macroscopic dissection of the isolated petrous bone. Until the advent of petrous bone microscopy the method was used mainly for exercises in anatomy and surgery. "Microdissection" as revived lately by Neubert (1950) tends to retrieve the "lost dimension."

Schalle (1877) criticized the methods of removal of the organ of hearing with the epipharynx and associated soft parts. He found the methods published prior to his time unsatisfactory, particularly those of von Tröltsch, Voltolini, Lucae, Zaufal, Luschka and Wendt. His own method in which he paid full attention to the "piety of suspicisious onlookers" was much too complicated to find followers. He complained that only a few physicians had a clear-cut picture of these hidden organs based on direct inspection.

von Tröltsch (1881) sawed along two lines, one somewhat behind the mastoid process and the other across the lesser wings of the sphenoid and the center of the zygomatic process. Both cuts penetrated the soft tissues beneath the cranial base.

Politzer, as quoted by earlier authors, used two saw cuts, one behind the mastoid process, which ended in the midline of the clivus, and another from the center of the zygomatic process to the tubercle of the sella. In 1889 he gave detailed instructions for the use of gouge and mallet in cases where only one of the temporal bones was wanted, and this without adjacent parts of the eustachian tube or pharynx. He used mainly a gouge with a straight cutting edge of 3 cm to remove the organ of hearing "in a very simple and quick way." His pentagon, five lines for penetration with the chisel, might be considered classic (Fig. 1). They were probably used most frequenty and universally.

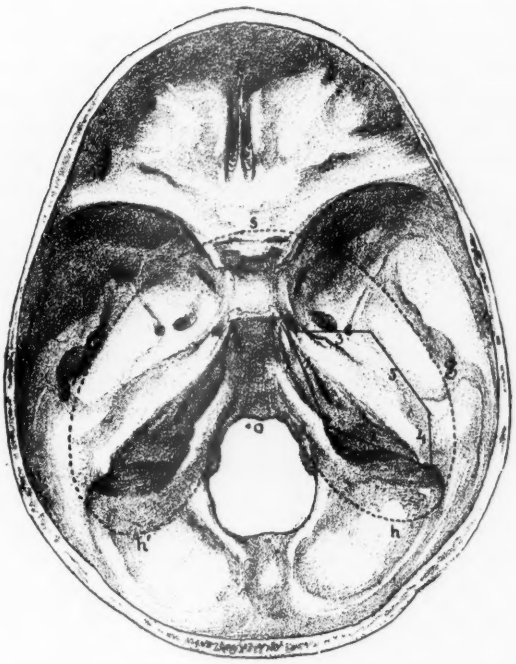


Fig. 1.—Politzer's illustration (1889).

Siebenmann (1897) used a saw to remove a block which contained the parts desired for examination.

Manasse (1917) advocated no further touching of the petrous bone with gouge or saw after its removal prior to decalcification.

Alexander and Fischer (1925) rejected categorically all methods which include work with the gouge.

Eckert-Möbius (1926) described the technique used by the school of Wittmaack as two saw cuts diverging in their downward progress. One saw cut placed immediately in front of the petrous bone ended medially in the foramen ovale. The other saw cut placed immediately behind the petrous bone ended medially in the foramen magnum. The bony ridge between the tip of the pyramid and the bodies of the sphenoid and occipital were severed with a chisel. The soft tissues at the base were cut with knife and scissors.

Dorothy Wolff (1947) advocated the use of the Albee osteotome (Jetter and Scherer B-2653), and a chisel with a thin, broad

blade of 40 mm. The blade drove readily into the petrosa. For infant skulls she found a narrower blade advantageous. Four planes of cleavage, described in detail, were made with the chisel. Care was taken never to complete a single incision at a single blow from the hammer. The cuts were mapped out with light strokes and repeatedly deepened until the block loosened in its bed. Then scissors were used to cut the internal carotid artery, the palatine muscles, the eustachian tube and the cervical fascia.

Finally, Guild (1948) summarized his experience in these techniques. All major cuts should be made with a saw instead of with a chisel. A rocker-type saw is found generally around the autopsy room. Preference is given to saw driven by the shaft of a small motor, an electric drill with a so-called pistol grip, the chuck of which has been adapted to hold a short rod carrying the saw blade. Four saw cuts were described in detail. Removal of the block outlined by the four saw cuts is often the most difficult part of the entire procedure. With proper tools and a bit of experience, removal of both temporal bone blocks in good condition can be accomplished in about 20 minutes after the brain has been removed.

The gouge and saw each have advantages and drawbacks, and consequently each found friend and foe. The gouge can be directed at any moment in a desired direction. However, as Wendt and von Tröltsch warned, fissures might be produced in parts where they were least wanted. Without the necessary circumspection, said Politzer, "these fissures of the bone will run in every direction, resulting in laceration of the drum, in dislocation of the ossicles, and further damage." Guild warned of extensive fracturing of the bone caused by the wedge of the chisel. Alexander and Fischer unconditionally rejected the gouge. In their opinion the rongeur, too, will produce direct injuries such as fissures, and indirect ones by shattering when the bone is pried off. Moreover, laceration of thin membranes and dislocation of freely movable nerve branches will be produced. Encountering these breaks in continuity later in microscopic preparations will add to the difficulty in interpretation of the origin of fissures seen in so many temporal bones (Kelemen).

As Alexander and Fischer pointed out, the saw produces a bone pulp which clogs the openings of the intrapetrosal spaces preventing penetration of fixing fluids. According to Dorothy Wolff the electric saw is very difficult to control and its use necessitates wearing a mask by prosector and observers because contaminated bone dust may be thrown into the air.

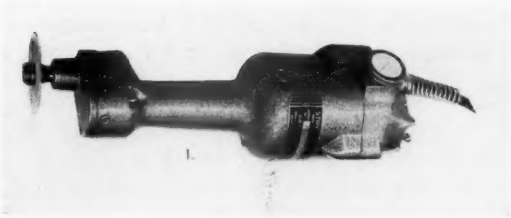


Fig. 2.—1. Handle containing motor.

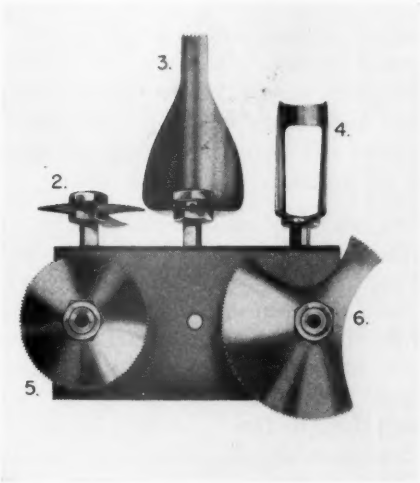


Fig. 3.—2. Small sectioned blade; 3. end cutting blade; 4. bone scoop; 5. circular blade; 6. large sectioned blade.

Gyergyay (1930) constructed an instrument for the extraction of the organ of hearing, eustachian tube, parts of the nose and nasopharynx in a block without mutilation of the cadaver. This instrument consisted of a tube serrated at one end like a trepan and of such diameter as would encompass the petrous pyramid. The instrument was driven from the outside of the skull, at a proper angle, against the midline of the cranial bone. When withdrawn it contained the desired parts.

The need of such an appliance was corroborated by Beck (1938) when, apparently unaware of the previous communication, he described his "temporal bone corer." This instrument was, according

to Beck, first specified by F. Lowell. In a modification, an extractor was added to a trephine 9.5 cm long of 5 cm diameter. The device was applied through a simple incision over the mastoid.

Recently an instrument has been constructed which seems most suitable for the removal of the petrous bone or any desired bony block. This is the autopsy saw of Stryker (Figs. 2 and 3). It operates on a small motor as a handle, is electrically powered, and uses either AC or DC. Instead of rotation, the principle is high speed oscillation. The blade is arborescent, has alternately offset teeth, and cuts on forward and backward strokes. Of the several types of blades offered, two can be used best for the removal of the petrous bone. The first is an arborescent sectional blade, with a broad segment for linear cuts and a narrow segment for cutting the ends. The second is a bone scoop for separating the petrous tip. The sectional arborescent blades permit cutting of the bone in depth. Since the blade does not rotate, no material is hurled and blood and infectious matter is not flung into the face of the operator.

The following procedure has been satisfactory: If the body was to be displayed, the head and upper body was first embalmed through the intact aortic arch with 10 per cent formaldehyde solution arterial injection. The skull was then opened by interlocking saw cuts and the brain removed. The dura of the cranial base was left intact and the outline of the desired block carefully delineated on the dura with a sharp knife. The broad blade of the saw was then applied along the pyramid, first in front and then behind it. The size of the block was determined by the individual case. The petrous tip was separated with the narrow blade or the bone scoop. The block was loosened by gentle prying, not hammering, with a large wide chisel. The now accessible carotid artery and facial nerve were cut with long curved scissors. The remaining soft parts were cut with long curved Mayo type scissors and long handle type Bard-Parker knife. The block, covered with its dura, was gently lifted from the cranial base on the blade of the large chisel. Following the directions of Manasse to avoid unnecessary damage after removal, no further cutting of the block was attempted until at least partial decalcification was accomplished. The factor of time which has been reduced by every efficient method to a minimum, was negligible with this procedure.

After a century, the end of endeavors to obtain undamaged specimens of the organ of hearing is not in sight. Doubtless, more and more accomplished techniques will emerge; one of these well might be the ultrasonic which is designed to penetrate bone and soft

tissue with the least possible destruction. Electrophoretic decalcification may shorten the long waiting period until the excised petrous pyramid appears under the microscope. In any event shorter and simpler technical procedures will help to bring pathology of the human temporal bone out of its present impasse. This impasse lies in the competent interpretation of temporal bone pathology as the province of the comparatively few.

SUMMARY

The one hundred year history of the endeavor to remove the petrous bone quickly and undamaged from the cranial base, for macro- and microscopical investigation is sketched. Application of a new device is suggested.

The suggestions of Dr. Jack Mickley, Department of Legal Medicine, Harvard Medical School, in preparing this paper are gratefully acknowledged.

414 BEACON STREET.

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# Scientific Papers of the American Laryngological Association

XL

## THE ADDRESS OF THE PRESIDENT

H. Marshall Taylor, M.D.

Jacksonville, Fla.

It is with profound humility that I express to you my genuine appreciation of the honor you have conferred upon me by elevating me to the presidency of this Association. One who pauses to reflect, as have I, upon the galaxy of illustrious members who have preceded me in this office, could not feel otherwise.

When we speak of science in this advanced day we recognize that it has not changed any of the real laws of nature; but we know it has fathomed many of nature's intricate secrets. If we had no science, nature would present the same phenomena as at present. The heavenly bodies would move with equal regularity and preserve the same relative positions although no system of astronomy had been formed. The laws of gravity and of motion would operate in the same manner as at present if we had no such science as natural philosophy. Before the science of chemistry existed, the affinities of substances for each other were the same as they are now. It is an important truth, and one which cannot be too much impressed upon the human mind in all scientific investigations, that no effort on the part of man can change the laws of nature.

Nevertheless, by the faculties of the mind with which God has endowed us, we are able to examine the properties of the materials surrounding us. Sometimes we can offer opposing forces which retard and alter disease processes, thereby relieving suffering and prolonging life.

It is natural to suppose that the various forms of bacteria had relatively the same virulence and toxicity in the days of the Cro-Magnon man some 20,000 years ago, and in the time of Hippocrates and Galen, as they have today. Yet these poisonous enemies of mankind now have been robbed of much of their disease-producing power by medical science through the wealth of scientific investigation carried on in the modern laboratory by the bacteriologist, the pathologist and the cytologist. Likewise, the cancer cell most likely must have possessed some of its present characteristics in the early history of human life. Sometimes, however, much of its malignancy has been altered or thwarted by the discovery of the roentgen ray and the splitting of the atom.

The scientist now tells us that 150,000,000 atoms in juxtaposition may measure only one inch. Furthermore, he avers that he can isolate this infinitesimal atom, divide it into its component protons, neutrons and electrons and measure the velocity of the electrons, which may exceed 100,000 miles a second. With this sobering information in mind, who has the temerity to predict the ultimate service to humanity of the beta and gamm rays of radium and the radio active isotopes, for example?

All these facts make us realize that the physician of today lives in an exciting and almost a magic world, with more and more avenues of research constantly being opened to students of medical science. With the phenomenal development of its various branches, where is medicine leading us today? While I am aware that I am speaking to a group of physicians primarily interested in rhinology and laryngology, I am convinced that all of us realize the term specialist properly becomes almost obsolete in some particulars.

Nevertheless, we contemplate the future of our particular branch of medicine with special interest and concern. We may think of it as one of the younger branches. Yet, we are told by the medical historians, Stevenson and Guthrie, in their recent "History of Oto-Laryngology" that the earliest physician known to us by name was a rhinologist. He was Sekhet'enanch, the medical attendant of a Pharoh of about 3500 B. C. A limestone slab with a drawing of the physician was found in the tomb of the King bearing the inscription, "He healed the King's nostrils." Were we to speculate on the sucessful therapy employed by this early Egyptian rhinologist, how many of us would surmise that he advised the Pharoh to discontinue the use of nose drops?

As we review the past, the advances made in recent times, many of them by members of this Association, make us take stock of the present as we look ahead to greater achievements.

Like other sciences, the science of medicine has grown through the ages as a result of cumulative knowledge. But for Archimedes, Galileo, Roger Bacon, Newton and Descartes with their conspicuous achievements in the realm of synthetic thinking, Einstein would not have evolved his theory of relatively. Such acts of synthesis are essential to the upbuilding of the scientific process. Thinking along these lines some 2,000 years ago, Hippocrates, when asked "Who is the physician that is an honor to his profession?" replied:

> He who has merited the esteem and confidence of the public by profound knowledge, who has been led through the whole circle of sciences, who moreover neglects not to observe all the causes that may produce disorder in the animal economy.

Adam Politzer, known as the Father of Otology, once said that the essence of medicine could be summed up in these words, "Everything is connected with everything." In his presidential address before this Association in 1920, Dr. Harris P. Mosher observed, "A specialist should forage in every field of medical activity." Such expressions as these, coming from the pen of such savants, should stimulate every physician to keep his eyes and ears open and his mind alert. They serve to remind us of that sense in which the term specialist should ever be outmoded.

Recently, as a subject of discussion the future of otolaryngology has gained great popularity. More and more frequently it seems to find a place on the program of our medical meetings. In fact, it has become so much the vogue as to be almost a fad or a fashion. Unfortunately, some authors and editors have portrayed otolaryngology as "a shrinking specialty," subject to encroachment and erosion from other branches of medicine. The tenor of a recent editorial in a leading medical journal almost intimates that rigor mortis is setting in. If this fad does not soon run its course and this high degree of pessimism is allowed to grow, we may soon be hearing derogatory remarks from radio commentators and seeing disparaging presentations on television. Unless we take aggressive steps to arrest this trend, who knows but that we may yet be confronted with Milton Berle or Humphrey Bogart impersonating the otolaryngologist as the dying gladiator?

Although widely publicized, these gloomy forecasts express only the opinion of the individual essayists and editors who make them. Their import, however, is unfortunate both for our teaching institutions and our special hospitals. Such propaganda invites a dearth of applicants and a consequent let-down in the training program. It follows that students and interns would forego a sufficient training period to make them competent, and the public would inevitably suffer. For the vociferous minority who seemingly delight to decry our branch of the healing art, let there be a more articulate majority to uphold it.

Even though the antibiotics have made less surgery necessary in some of the sinus and otitic infections, there nevertheless remains to the otolaryngologist today a wider field than many of the older members of the Association had when they began their professional career. It is my observation that the younger physicians now electing to cast with us their lot in medicine are working to capacity and utilizing their training in a broader field than many of their senior colleagues ever have done. Those who come from the best institutions with a background of internships and residences covering three or four years are not only well grounded in diseases of the ear, nose and throat but also versed in deep surgery of the neck, surgery of the salivary glands, bronchoesophagology, reconstructive surgery of the head and neck, allergy, audiology and physical medicine.

Certainly one is fascinated by the innumerable facets of the jewel of otolaryngologic research, especially so upon reading the "Forty Questions in Otolaryngology" posed by Dr. Arthur Proetz in his self-styled role of fool who asks questions wise men cannot answer (The Annals of Otology, Rhinology & Laryngology, September, 1951). As for the scope and practical aspects of the practice of otolaryngology, what thoughtful, fair-minded, informed analyst will gainsay its equally broad and intriguing appeal? Fritz Kahn has reminded us that in an opera an entire orchestra plays, but above it sings one human being with a small larynx, and the human voice triumphs over all artificial instruments. Likewise, the trained laryngologist and the skilled rhinologist will always transcend all others in the diagnosis and treatment of diseases of the upper respiratory passages. That is my considered judgment, in which I believe the members of this Association will heartily concur.

It has been said that it is very hard to press gold and diamonds and sapphires into the service of art, but the poor ball of clay which has been pinched by the fingers of Michelangelo may show the sign unmistakable and be treasured for centuries. And so it is with otolaryngology. Let us not lose the vision. Let us sharpen our perceptions. Let us search our hearts and minds and call forth the gift that is within us to mold the clay of our professional knowledge and experience into contributions that will leave the world better off. It is ours to participate in the synthesis of medical thinking which today ventures to cross the threshold of a new era.

Whither are Aesculapius and the savants of the ages leading us through the achievements of our modern day? In this dawning atomic age, who can visualize the future of the science and the art of medicine? Who indeed would narrow the concept of any branch of the healing art in this epoch of expansion? So great are the possibilities, so broad the concepts, that we may well be mindful of St. Augustine, who centuries ago sought to answer the question, "What was God doing before He made heaven and earth?" Wrote the eminent philosopher and divine, "He was making the hell for the persons who ask that kind of questions."

The questions which yet confound the wise men of otolaryngology are both fascinating and stimulating - and not unanswerable. The quest for the solution to the many problems assures and makes it bright. The Golden Age of Otolaryngology lies ahead.

# XLI

# LARYNGEAL TUBERCULOSIS

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The purpose of this paper is to discuss briefly laryngeal tuberculosis, one of the most distressing complications of the disease, rather than to dwell exclusively on the accomplishments of streptomycin, which is the fashion at the moment in medical literature.

Pain in swallowing was formerly relieved by anesthetic lozenges, later by nerve block, while lactic acid locally, and the electric cautery, did away with much obstructive swelling, and silence gave local rest. Continuing pulmonary involvement led to continuing cough, while tracheal edema followed by stenosis caused still more interference with breathing, and thus further extension into uninvolved lung tissue.[11, 12]

George Fetterolf reported to this Association in 1914 the autopsy findings on patients dead of tuberculosis in Philadelphia; of these, 83 per cent showed laryngeal involvement.[1] Sir St. Clair Thomson, himself in youth a victim of the disease, in 1919 discussed the prognostic importance of laryngeal tuberculosis and the value of proper laryngeal examination; he looked upon the appearance of this complication, occurring in at least 22 per cent of all sanitarium cases, as most dangerous.[2] In 1937 he stated: "Tuberculosis of the larynx is particularly interesting on account of its frequency, gravity, and the importance it bears to prognosis."[3] But Ormerod has recently reported from London the results of a thirty year statistical study, showing a diminution from 25 per cent in 1921 to two and one-half per cent in 1939.[4]

Current opinion among American phthisiologists may be summarized about as follows: "It is difficult or impossible to obtain accurate statistics regarding the incidence of tubercular laryngitis. In the first place, the incidence seems to vary considerably in different parts of the country, being influenced no doubt by factors such as standard of living, nationality, race, education, etc. The incidence among admissions to tuberculosis institutions likewise varies according to the type of institution. For instance, some institutions will

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Presented before the American Laryngological Association, Toronto, May 23, 1952.

draw only from the higher economic levels. On the other hand many state and county institutions, especially in areas where there is a bed shortage, select patients for admission on the basis of their prospects for recovery, their need or suitability for collapse therapy, surgery, etc. In institutions established primarily for segregation and custodial care, with many terminal cases, the incidence is naturally high. Thirty years ago, tuberculous laryngitis occurred as a complication of far advanced pulmonary tuberculosis in perhaps fifteen per cent of cases. In moderately advanced pulmonary tuberculosis not more than two or three per cent were so complicated; in minimal pulmonary tuberculosis not more than two or three per cent. At present, tuberculous laryngitis is thought to occur as a complication in only about four per cent of all cases." [5, 11, 12]

How has this beneficent change come about?

Several factors have been at work, notably control of coughing, by phrenicotomy and by various types of lung surgery—collapse therapy, rib resection, and removal of hopelessly involved lung tissue.[11] Early recognition of the disease through chest plates has of course contributed much to recent therapy. More recently still, the use of streptomycin, often coupled with paraaminosalicylic acid, has in spite of dangers to the auditory and vestibular nerves cleared up throat lesions rapidly. But it is not necessarily curative for extensive pulmonary damage.[10-12]

Most of all, freedom from the rasping, tearing assault of sputum upon the interarytenoid space, vocal cords and epiglottis has done away with the implantation of virulent germs into these regions. Particularly, when patients became so weak that it was hard for them to expel sputum, thus permitting drying and crust formation, the eventual removal of such crusts would tear off masses of mucosal tissue and leave sensory nerves exposed. Granulomatous masses, often partially occluding the glottis, were materially reduced by Spencer's technique of electrocautery;[6] but the efforts involved in coughing often reinoculated such healing tissues. "Cauterization of tuberculous lesions of the larynx, pharynx, mouth, gums, etc. continues to hold an important place, but it is the only one of the older methods of treatment not displaced by the antibiotics. Of course, the tubercle bacillus soon becomes resistant to streptomycin, but less readily to the newer dihydrostreptomycin." . . . "We believe the antibiotics at first arrest the disease, and then cure it, except in long standing or late and advanced cases."[7-12]

However, long years before the use of streptomycin, attempts to put the injured lung at rest, or to remove its damaged or cavitated

portions, were successful.[5, 11] Thus coughing was cut down, and habituation to cough troches and narcotic medicines became unnecessary. Management of tracheal ulceration has been much improved by antibiotics, and the heavy stenotic scars resulting from strong silver nitrate or actual cautery are now rarely seen.

Chest films by public health services are now screening out many early cases which formerly became severe before coming to treatment. Use of BCG vaccine prophylaxis has, in Europe, Mexico, South America and Japan, apparently cut down the incidence of juvenile tuberculosis, and many epidemiologists hope it may soon be generally employed in the United States and Canada. Economic conditions, the privations of war, lack of essential protein food, all play a part in continued susceptibility to pulmonary tuberculosis.[4]

Nowadays, younger laryngologists, unless they have had residences in sanatoria, may never have seen a case of tuberculosis of the larynx; and the average practitioner rarely sees one in private practice.

Like diphtheria, it is a disappearing disease.

In the incidence of tubercular laryngitis, the reduction of 83 per cent in 1914 to some two per cent today is one of the outstanding achievements of modern medicine; and we may feel that one of the severest complications of tuberculosis has been practically wiped out, thanks to the control of coughing, and, more recently to streptomycin and to certain less toxic but potent drugs still under test, such as isoniacin hydrazide.

No older laryngologist will ever forget these pitiful patients who followed him with pleading eyes in clinics and sanatoria, asking voicelessly for relief of pain and difficulty in breathing.

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# XLII

# BRANCHIOGENIC CARCINOMA

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AND

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At the 1946 meeting of the American Laryngological Association Blassingame[1] presented an excellent paper on "Congenital Cysts and Fistulae About the Head and Neck." Blassingame devoted considerable attention to the work of Winglowski who opposed the generally accepted theory of the branchial origin of these cysts and stated that these were more likely to be remnants of the pharyngothymic duct formed by the "anlage" of the thymus in its descent to the post-sternal space.

However, the term, branchiogenic cyst, by long usage, is generally accepted as applied to these cervical cysts of embryonic derivation. Of even more controversial nature than their exact origin would seem to be the question as to whether these cysts ever became primary sites of malignant changes. Since Lorenz in 1913 applied the term branchiogenic carcinoma to the condition first described by Von Volkman in 1882, this diagnosis has appeared from time to time in the literature and most text-books in pathology devote at least reference to it. Moore[2] states that epidermoid carcinomas are associated with branchiogenic cysts in many cases and "there seems no doubt that a carcinoma may take origin from the epithelium of a branchiogenic cyst. The presence of numerous lymphoid follicles serves as histologic evidence of their origin."

Crile and Kearns,[3] in 1935, stated that this was a distinct pathological entity, although the mode of origin was obscure. While they found fewer than 100 cases reported in the literature they added 28 of their own. They described the clinical picture, stating it was one of rather rapid growth, that the tumor was adherent to the deeper structures of the neck but not to the skin, and that there might be involvement of adjacent cranial nerves as well as the cervical

sympathetics. Pain was sometimes referred to the face, occiput, base of the tongue, shoulder, or arm. The differential diagnosis included tuberculosis, Hodgkins disease, mixed tumor, lipoma, lymphadenitis, cystic hygroma, hemangioma, and aneurysm. The pathological findings were those of squamous cell carcinoma together with evidence of a cyst wall and often a definite capsule infiltrated by tumor tissue. In their opinion the pathogenesis was an embryonic rest stimulated to neoplastic activity. While the prognosis was poor they felt that a combination of surgery and irradiation offered the best results.

Hanover and Clark[4] used the terms "adenolymphoma (papillary cystadenoma lymphomatosum) or branchioma" and stated that origin from rests of branchiogenetic entoderm or heterotopic rests of eustachian tube or pharyngeal entoderm was the most tenable theory.

Jackson[5] states that "branchiogenic fistulous tracts may occur and undergo malignant change."

In 1949 Ward, Hendred and Chambers[6] in a study of branchiogenic anomalies reported seven cases of branchiogenic carcinoma, three of which showed histologic evidence of originating in the cyst wall. They stated that the microscopic findings were those of squamous cell carcinoma together with a cyst wall thickened and infiltrated with tumor cells or, if small, almost entirely replaced with tumor. The tumor cells appeared arranged in whorls or branching cords with very little connective tissue stroma. Cell nests with pearl formations could be noted. Usually the tumor was undifferentiated.

On the other hand Martin[7] in 1948, while granting the theoretical possibility of cancer arising in a branchiogenic cyst, stated that the diagnosis was rarely justifiable and that the condition was more likely to be secondary to a silent primary lesion in the pharynx or larynx. He doubted the validity of the diagnosis in the cases reported, saying that he had found no definite evidence that any such specific tumor existed and that the only acceptable proof of such would be the demonstration of cancer actually arising in the wall of a branchiogenic cyst.

In a later communication[8] published in 1950, out of some 250 cases reported by 34 different authors, Martin found only three, in a series of 80 reported by Oliver[9] from Johns Hopkins Laboratory of Surgical Pathology, which presented reasonable evidence of branchio-

genic malignancy. He stated that such a diagnosis must rest upon the assumption that there was no primary lesion elsewhere; that frequently the first, and for sometime the only, manifestation of a silent malignant tumor in the mouth, nasopharynx, pharynx, larynx or even below the clavicle, was a metastasis noted in the neck. If this occurred as a pathological entity he felt it should be found in the embryologically similar thyro-glossal duct cyst, yet no such cases had been reported. Commenting upon the difficulty in pathologically differentiating between metastatic and branchiogenic carcinoma, he stated that a definite diagnosis could not be made on a histologic basis and that the actual existence of branchiogenic carcinoma as a clinical entity was hypothetical.

It is rather difficult to reconcile these conflicting views. Obviously the clinical and microscopic findings in the cases reported confirm the presence of both branchiogenic cyst and carcinomatous tissue. It is then purely a question as to the point of origin of the malignant process. Is it primary branchiogenic carcinoma, or metastatic carcinoma coincident with branchiogenic cyst? While at first hand this question of origin might seem rather academic, inasmuch as the immediate management would be similar, it becomes extremely important when one considers the possibility of an overlooked or silent primary lesion elsewhere.

Further pursuing this question one might consider whether a metastatic process in the neck could invade the branchiogenic cyst, or conversely, could a primary lesion within the cyts itself extend to surrounding tissues, even conceivably into the tonsillar fossa.

The following case is reported—not as sugesting any solution to this controversy but merely for purposes of discussion:

### REPORT OF A CASE

F. D., male, aged 39, was seen with a history of a tumor of the neck of some five months' duration. Examination of nose, nasopharynx, pharynx, and larynx was entirely negative. He had small tonsils which appeared normal. Operation revealed a characteristic cyst 4 cm in diameter with a stalk leading upward and apparently becoming obliterated in the region of the muscular bed of the tonsillar fossa.

The pathological report was as follows: "Sinus-like structure lined by papillary processes covered with stratified squamous and

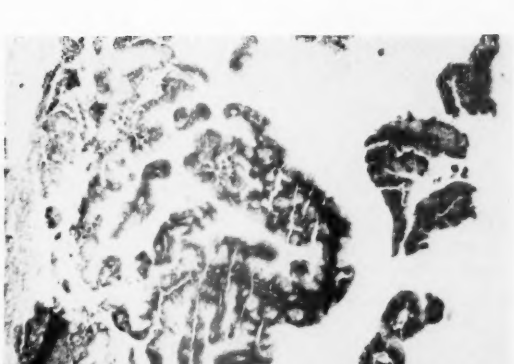

Fig. 1.—Specimen from last surgical procedure, showing papillary nature of the tumor.

transitional epithelium-marked lymphoid stroma. Wall dense and fibrous. Some areas show granulation tissue with cellular infiltration suggesting frequent or long-standing infection. Large collections of fat-laden macrophages in the walls. No evidence of malignancy. Diagnosis: Branchiogenic Cyst—Chronic Inflammation."

The patient made an apparently uneventful recovery and was free of symptoms for 13 months. Then he noted a small swelling just below the scar of the previous operation. This had increased in size slowly when seen eight months later, or 21 months after operation. He was readmitted to the hospital where operation revealed a cystic tumor apparently similar to that previously removed but without a demonstrable stalk. This was markedly adherent to the underlying tissues making it necessary to ligate and resect a portion of the internal jugular vein.

The pathological report was as follows: "A cystic structure lined by transitional epithelium with suggestion of stratified epithelium in some areas—somewhat papillary type of architecture. Minimal anaplasia of some groups of cells. Diagnosis: Branchiogenic Cyst.

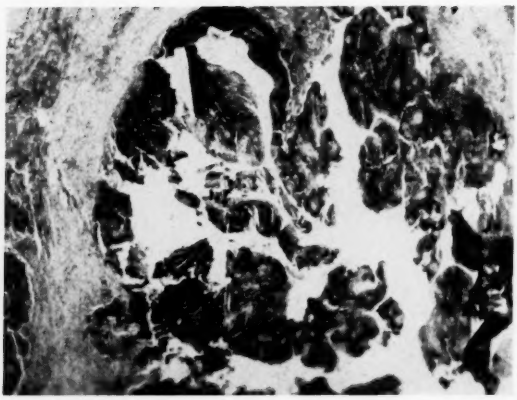

Fig. 2.—Specimen from last surgical procedure, showing papillary architecture, epithelial hyperplasia, and, at the top of the field, invasive tumor and lymphoid tissue.

Again the convalescence was apparently uneventful. Three months later, however, the patient reported, showing a large indurated tumor posterior to the original site and presenting under the margin of the sternomastoid muscle. Biopsy report was "branchiogenic cyst."

Employing an inverted T incision as advocated by Mosher, a large indurated tumor surrounded by inflammatory tissue was found underneath the sternomastoid muscle. Following operation the patient developed a right recurrent laryngeal paralysis which persisted for 17 days before mobility was restored.

The pathological report at first was returned as branchiogenic cyst but careful re-examination of serial sections revealed nests of anaplastic cells and the diagnosis was changed to "epidermoid carcinoma, grade II-III, arising in a branchial cyst."

Deep roentgen therapy was now instituted. The patient seemed again to be making satisfactory progress when ten months later, or 39 months after first noting the presence of the cyst and 35 months after the first operation, a small neoplastic-appearing mass was noted in the lower portion of the right tonsillar fossa. Biopsy reported was

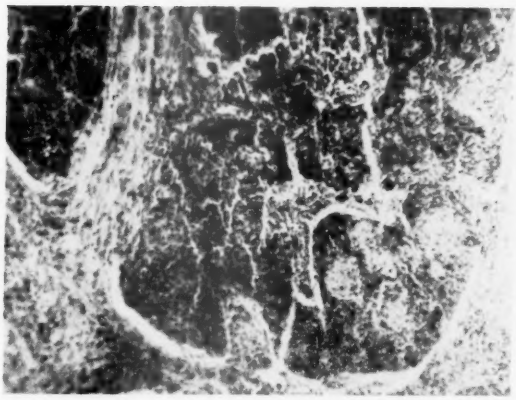

Fig. 3.—Biopsy of tonsil, showing epidermoid carcinoma growing in sheets, with epithelial pearls and some necrosis.

epidermoid carcinoma, grade III-IV. Interstitial irradiation, with a dosage of 3300 mc hours, was combined with a second course of external roentgen therapy. This resulted in complete regression of the pharyngeal tumor and to date (some two years now) there has been no evidence of recurrence in the neck.

The original specimen in this case was the only one which showed the characteristic architecture of a branchial cleft cyst. The epithelium, mainly stratified squamous in type, overlying active lymphoid tissue, with a scanty fibrous wall, provides incontrovertible evidence of the type of lesion involved. Subsequent specimens, while suggestive of this type of tissue, showed sufficient distortion of architecture as to be inconclusive in themselves. The last operative procedure produced a mass of tissue which grossly was not demonstrably cystic, but which microscopically showed a distinctly cystic architecture with papillary masses of epithelium which was definitely anaplastic and rich in mitotic activity. The stroma was fibrous, and showed pronounced lymphocytic infiltration, with some areas actually suggesting lymphoid tissue. This appearance is certainly suggestive of residual branchial tissue, or possibly, an attempt on the part of the tumor to form tissue resembling that of the respiratory tract.

The question of possible origin of carcinoma in a pre-existing branchial cleft cyst has been argued on both sides for some time. There is no denying the fact that all tissues of the body are susceptible to the development of malignancy in some degree. Many developmental anomalies show an even greater tendency to this type of change. Although it is true that branchial cleft cysts are a common site of lymph node metastasis from lesions of the mouth and throat, the demonstration of characteristic branchial cleft architecture on original examination certainly suggests consideration of this structure as the site of origin. The fact that the tumor recurred and finally involved the tonsillar area after three years, is still consistent with direct extension, especially since a silent carcinoma of the tonsil with spread to the neck would be expected to have involved additional nodes after this length of time. The occurrence of a primary carcinoma of the tonsil in a 39 year old man is also sufficiently unusual as to add weight to the opinion that the origin of the tumor was in some other area.

Many findings in this case are difficult to satisfactorily explain. The first operation revealed what was apparently a typical branchiogenic cyst except, possibly, for the histologic finding suggesting chronic inflammation. The apparent obliteration of the stalk distal to the tonsillar fossa may have been due to a technical error, resulting in leaving branchial tissue in that region. But the cyst proper was clearly developed and apparently removed, and the recurrence was below the level of the original incision and was not evident for over a year. Was the microscopic finding of minimal anaplasia in the second specimen of any significance? The third operation disclosed definitely carcinomatous tissue. Was the neoplasm which later made its appearance in the tonsillar region a primary lesion, long silent, or an extension from the pharyngeal end of the stalk? Removal of the tonsil and inversion of the stalk into the pharynx would have been advisable in the light of subsequent developments, although many authorities, including Lahey,[10] no longer feel that this is necessary and it has not been the writer's practice to do so routinely.

The diagnosis of branchiogenic carcinoma was accepted as the best explanation of the recurrence of the tumor. At the same time we must admit the possibility of technical error as in both earlier operations we were limited by the type of incision commonly em-

ployed for the removal of branchiogenic cysts and only at the last, with the inverted T incision, was there really adequate exposure.

Obviously we cannot definitely determine in our case the primary site of the carcinoma. Considering the sequence of events, however, two possibilities come to mind. Was this the result of metastases from a silent neoplasm in the tonsillar fossa invading branchial tissue inadvertently left at the first operation? Or could embryonic tissue remaining in the pharyngeal end of the stalk, stimulated by chronic inflammation develop malignancy with the eventual appearance of neoplastic tissue in the pharynx? The first would seem in accord with Martin's views and, considering his extensive experience, should not be discarded, although rather at variance with our operative findings. At the same time it is interesting to speculate upon the second possibility, which would lend weight to the views of those who consider branchiogenic carcinoma a distinct pathological entity.

## COMMENT

Reviewing our experience with the case, several points of practical value are suggested:

1. The necessity of wide exposure of the operative field in any case showing adherence to underlying tissue, or in which there is recurrence of the tumor.

2. The importance of serial section study in all suspected cases. Reliance on biopsies may be misleading.

3. Inversion of the stalk, at least in certain cases.

4. As so forcibly brought out by Martin, the persistent search for a silent primary lesion in all maligant manifestations in the neck.

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XLIII

# THE SUPRAHYOID APPROACH TO SURGICAL LESIONS AT THE BASE OF THE TONGUE

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The feasibility of employing a suprahyoid approach to tumors situated on the base of the tongue has long been recognized. The anatomist, Vidal de Cassis,[1] in 1826, was the first to suggest an incision of the anterior wall of the pharynx as being the proper surgical approach to lesions in this area.

Malgaigne,[2] in 1834, recognized that the entrance to the hypopharyngeal space by an incision through the anterior wall of that space immediately above the hyoid bone should be a distinct advantage in dealing with lesions situated on the base of the tongue and the tip of the epiglottis. His deductions were based on studies of autopsy material. It was not until the latter part of the nineteenth Century however, that the anterior pharyngotomy approach was employed for tumors inside the pharynx. It would appear, from the literature on the subject, that the surgeons who first performed transverse pharyngotomies obtained their cues from observations of would-be suicides.

Jeremitsch,[3] in 1895, reported an attempted suicide in which the cut, above the hyoid bone, was a true suprahyoid pharyngotomy. His description of the wound was as follows: "On the front of the neck, about 2 mm over the tongue bone and parallel with it, the cut is 12 cm, reaching the back part of the pharynx, it stretches from one nodding muscle to the other, gaps widely but shows clean edges. When he coughs and throws his head backward, the injury gaps still more widely and the uninjured epiglottis rises. In both corners of the injury we could see the inwardly situated jugular veins. No hemorrhage." So impressed was Jeremitsch with the findings in this suicidal case that he declared his intention of performing all future operations in the pharyngeal cavity in the area of the root of the tongue with the same type of incision as used by the suicide.

Grünwald,[4] another influential writer of that period, observed that the suicides had been the teachers of the surgeons regarding transverse pharyngotomy. The experience gained from such cases, he said, may lead to professional application of the principles of suprahyoid pharyngotomy.

Jeremitsch emphasized two significant facts which were outstanding in his case: First, bleeding was minimum. This observation can be well understood, since in the suprahyoid incision no large blood vessels are encountered in the site of the incision. Parenthetically, neither are nerves encountered. Second the favorable disposition of neck wounds to heal. He quoted from the literature and cited statistics from Billroth's Manual of General Surgery which supported this thesis. This favorable tendency for neck wounds to heal was also observed by Thompson[5] on soldiers whom he attended in English hospitals following the battle of Waterloo.

Dieffenbach[6] emphasized that injuries on the neck, committed by a second person, healed quickly without complications.

Our purpose in reciting these data, some of which may at first appear irrelevant, is to emphasize that there was an early recognition of the apparent suitability of the transverse pharyngotomy approach to the pharynx. The operation, in spite of its merits, soon passed into eclipse and was little heard of until very recent times. This decline in the popularity of the operation was all the more strange since the suprahyoid pharyngotomy gives the most satisfactory approach to tumors at the base of the skull, in the area of the epipharynx, and to tumors of the choanal regions and on the posterior surface of the soft palate.

Hofmann,[7] in a paper before the German Surgical Society in 1907, gave a comprehensive survey of surgical procedures dealing with tumors of the nasopharynx. He concluded that "the suprahyoid pharyngotomy proves excellent for observing the operative field in the nasopharynx. Since the walls of the pharynx are extremely extendable one can, by using retractors, pull the walls of the pharynx aside and the operation may be performed under the steady eye of the surgeon." He showed that by splitting the soft and hard palate in combination with the suprahyoid pharyngotomy, a broad approach can be easily obtained. This he contrasted sharply with the very bloody approaches of the other methods, such as, the Kocher[8]

method, the method used at the Bergman Clinic,[9] Lagenbeck's[10] Hertle's[11] and Garre's.[12] In evaluating the magnitude of the operation, he stated, since suprahyoid pharyngotomy involves no bone surgery, and does not cause much bleeding, one may consider it a minor operation. On the other hand, from the broad view of the operative field, one must judge it as radical as the other methods. Hofmann pointed out that, anatomically, the distance from the roof of the pharynx to the opening in the suprahyoid incision is no greater than that characterizing the Kocher operation where the septum and nasal cartilages are always in the surgeon's way. Also, when compared with Kronlein's[13] experiments using the lateral pharyngotomy, the superiority of the symmetrical approach to the epipharynx was clearly apparent. Even in the Lagenbeck operation with the resection of the lower jaw added to a lateral pharyngotomy, the same free look into the epipharynx was not obtained. The technique described by Hofmann for the suprahyoid pharyngotomy is as follows:

"The head hangs comfortably over the end of the table. An incision is made above the hyoid bone 12 cm in length, dividing the skin, platysma and fascia. Then the mylohyoid and geniohyoid muscles are detached and drawn upward from the hyoid bone. These muscles, together with the medial parts of the hyoglossal muscle, are bisected near the hyoid bone, enough of the ends of the muscles being left to help join them together after the operation is performed. Both submaxillary glands and the hypoglossal nerves are pressed backward. The mucous membrane of the pharynx is then incised, the length of the slash being relatively small."

An extended discussion concerning the bisection and reuniting the median raphe of the mylohyoid muscle is also included in this article. Continuing his description, Hofmann says that by pulling on the end of the tongue or with the use of a proper retractor applied to the severed root of the tongue because of the extensibility of the pharyngeal walls, an excellent view is obtained, especially of the nasopharynx. If one also uses a wide hook to pull the soft palate forward at the same time one can look unhampered over the whole of the nasopharynx, including the upper part of the spine and the lateral walls. One also has a good survey of the pharyngeal and faucial tonsils and the openings of the eustachian tubes.

Spizharniy[14] did the suprahyoid operation and reported on his results in 1906. From his experience he concluded that a combination of the suprahyoid approach with a buccal incision would give an excellent and unblocked approach to the operative field. He

admonished that the lateral segments of the hyoglossus muscle be left uncut and enough of the muscle stump be preserved so that it may be sutured afterwards. He stressed that it is necessary to locate and unite securely the cut ends of the median raphe. His careful observation of these important points in technique undoubtedly accounted for his excellent results.

Grünwald,[4] who performed the first successful suprahyoid pharyngotomy reported six cases in 1906. His results were unsatisfactory and it was due to his adverse criticism that the operation subsequently fell into disuse and little or nothing was heard of it until recent years. He insisted that, although the suprahyoid pharyngotomy furnished a splendid view of the base of the tongue, the epiglottis and faucial pillars, the end results were highly unsatisfactory. His criticism was based on one outstanding defect resulting from the operation, namely, sagging of the larynx. This unfortunate result, although in marked contrast with the experiences of Spizharnyi,[14] Jeremitsch, Hofmann, and others who reported satisfactory results in their operations, nevertheless influenced Grünwald to raise his editorial voice against the operation. The superiority of the pen over the scalpel in that instance resulted in placing this operation in the low historical niche which it has occupied for a half century.

In the years following, other methods of outstanding merit for entering the hypopharynx have been developed and successfully used in dealing with a great variety of lesions in the hypopharynx. Trotter's[15] operation for entering the hypopharynx laterally, so outstandingly employed by Orton[16] and others; the peroral approach by means of the suspension technique as practiced by LeJeune,[17] Simpson[18] and others and the subhyoid approach are well adapted for specifically indicated procedures. Our own limited experience and observations lead us to believe that, despite its poor historical past, the suprahyoid approach is the method of choice for certain lesions situated on the base of the tongue. A new method of transverse pharyngotomy combining the basic features of both the suprahyoid and subhyoid operations, has recently been proposed by A. Rethi of Budapest. No doubt, this operation will also have merit in some specifically indicated procedures.

With the use of antibiotics to prevent infection, combined with layer suturing of the involved muscles with particular attention to the reuniting of the ends of the median raphe of the mylohyoid, I

am firm in the belief that Grünwald's objection to the operation, namely, that it results in a sagging of the larynx, is no longer valid. My interest in this operation was inspired by a recent case, a report of which I submit:

REPORT OF A CASE

A white male, 86 years of age, was admitted to John Gaston Hospital August 30, 1950, having a very large tumor which completely filled his oropharynx. It had a smooth surface, was soft in consistency and was movable. It was attached to the base of the tongue by a pedicle 1.5 cm in diameter, lateral to the tip of the epiglottis on the left side. Dysphagia and dysphonia were present to a marked degree. His general condition was remarkably good for a man of his age. The Kahn reaction was negative.

A lateral roentgenogram showed a soft tissue mass encroaching on the air passage and displacing the hyoid bone downward. There was no evidence of invasion of soft tissue or bone. Biopsies were done on two separte occasions and they were reported, the first as lymphoid tissue and the second as chronic inflammation. On the ninth day after admission, the tumor was removed employing the suprahyoid pharyngotomy approach. Nitrous oxide anesthesia was used, being administered through the stoma of a preliminary tracheotomy. The hypopharynx was sealed off from the larynx with gauze packing. The initial incision was made one-fourth inch above the hyoid bone, longitudinally, from the median border of one sternomastoid muscle to that of the other, extending through the skin, platysma and deep fascia. The incision was then carried through the suprahyoid group of muscles including the anterior bellies of the digastrics, the mylohyoid, the geniohyoides and the central portion of the hyoglossus. The mucous membrane of the pharynx was then incised, the incision being easily controlled by placing the index finger of the supporting hand through the corner of the mouth and into the valleculae, as each was alternately entered. The tumor was easily delivered through the opening which was maintained by suitable retraction. It was then dissected from its base. The tonsil, on the corresponding side, being closely related to the tumor at its lower pole, was also removed by dissection. The defect in the base of the tongue was closed with interrupted chronic sutures and the bisected tissues by layer suturing. Particular atention was given to reuniting the severed ends of the raphe of the mylohyoid muscle since this, in

my judgment, is the most important support of the hyoid bone upon which the larynx and upper end of the esophagus depend for anchorage. The patient was fed through a Levine tube and given antibiotics. The wound healed by primary intention and the patient was discharged from the hospital on the 18th day at which time he swallowed without difficulty and had no disagreeable sequelae.

The pathological report on this tumor was as follows: The tumor was 6.5 cm in diameter. It was well circumscribed and presented ulcerations. It was friable and soft in consistency and yellowish-gray in color. Microscopically, a diagnosis of hyperplastic lymphoid tissue, with chronic inflammation, was made, although a low grade early lymphoma could not be excluded.

In our department at the John Gaston Hospital, four other patients have been operated upon in recent years for tumors on the base of the tongue, using the suprahyoid pharyngotomy approach. These patients were operated upon by Dr. Ralph Braund Chief Consultant in Tumor Surgery in the hospital. Two of these patients had adenocarcinoma of the posterior third of the tongue, one had an enormous lingual thyroid and the fourth had a hemangioma of the posterior third of the tongue. In each of these operations the accessibility to the site of the lesion, afforded by the suprahyoid approach, was most excellent.

These experiences and observations have encouraged us to believe that in this technique we have a valuable procedure in the surgical management of tumors of the tongue.

## SUMMARY

A historical review of the background of suprahyoid pharyngotomy as a means of approaching tumors on the base of the tongue is given.

The practicability of the operation is established as a means of approaching tumors in the epipharynx.

An unsatisfactory sequela of the operation, namely, sagging of the larynx occurred in the experiences of one man is mentioned. It appears from the literature that in his operations no attempt was made to suture the pharyngeal mucosa, nor to unite the ends of the sectioned muscles by suturing, the wound being left to heal by gran-

ulation. The influential writings of this one man discouraged the use of the operation for many years.

With the development of antibiotics to control infection and the assurance of the restoration of function of cut muscles by modern surgical technique, it is my belief that the objections to the operation which have formerly prevailed, can now be satisfactorily overcome. Suprahyoid pharyngotomy is an easy and satisfactory approach to the area of the root of the tongue and has a distinct value in dealing with specific surgical problems in this area.

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# XLIV

# RHINOLITH: A REPORT OF TWO CASES

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AND

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The discovery of a nasal calculus is an unusual and interesting experience. It is regarded as a rare condition but its occurrence may be more common than is generally supposed, for over 350 cases have been recorded to date and many have been encountered but not reported. It is also reasonable to assume that not infrequently small calculi form but become dislodged and ejected before reaching sufficient size to produce symptoms.

The pathogenesis of calculi in the nasal cavity is similar to that of calculus formation elsewhere in the body, such as the gall-bladder, the urinary-bladder, the kidneys, and the salivary glands. In these areas the concretions are largely endogenous in nature with a matrix composed of bacteria, leucocytes or tissue elements around which salts of magnesium, calcium, and sodium are deposited.

In the nasal cavity, the calculus is often formed around some small foreign body which has been introduced into the nose. These have been variously identified as shoe-buttons, bits of string, wads of paper, pencil erasers, beads, and other items. In one of our cases, the matrix was found to be a portion of a rubber band.

In most of the articles dealing with rhinolithiasis, the possibility of the endogenous nature of the growth is mentioned. As far as is known, this is purely theoretical and no evidence that these calculi may form around a matrix of dried pus or other endogenous material has been presented.

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One wonders why, if that were the case, so few rhinoliths are encountered considering the vast numbers of individuals with chronic sinusitis whose nasal and post-nasal spaces are seldom free of collections of purulent material.

Most of the foreign bodies found in a rhinolith are comparatively large. When none is seen it could be because the foreign body is too small or has lost its identity in the substance of the growth.

A possible nucleus for some of these concretions could be small particles of coal or stone dust or grains of sand. In silicosis, calcium collects around such particles trapped in the lungs and could well do the same in the nasal space.

Jarvis,[1] who has practised for many years in the granite centre of the country, told me that commonly patients show him concretions they have coughed up. He had not, however, observed any such collections in the nose.

Rhinoliths vary in size and shape, from small bodies to massive growths which entirely fill the nasal chambers and invade the adjacent structures. Many of the calculi reported are of good size and range between 50 gm and 80 gm in weight. The largest observed to date was reported in 1951 by Abu-Jaudeh.[2] It occurred in a 50 year old Arab whose symptoms dated back ten years. The calculus weighed 116 gm which was 6 gm heavier than the next largest. It produced distortion of the facial bones with a widening of the bridge of the nose, encroachment on the maxillary and ethmoid sinuses, and displacement of the right eye.

The signs and symptoms of a nasal calculus are those of a foreign body, to wit, a unilateral, malodorous nasal discharge associated with nasal obstruction. An occasional symptom is recurrent epistaxis which occurs as the growth erodes through the septum or the naso-antral wall. Most of the cases are of long duration with a gradual progression of annoying symptoms. In some there is no record of a previous nasal examination or medical care, while in others there is a history of exhaustive efforts on the part of the patient to obtain relief. In Gorman's[2] case the patient received treatment to his nose and ear two or three times a week for over a period of nine months without disturbance of the rhinolith which came to within one-half inch of the anterior naris and could be palpated on digital examination of the nasopharynx.

Rhinolithiasis must be differentiated from chronic maxillary sinusitis, bone necrosis, and malignancy, but the diagnosis is fairly well established when a probe or other instrument scrapes against a rough, hard surface with an unmistakable grating sound.

Treatment consists in removal of the rhinolith and when this is accomplished, the symptoms disappear. The large stones, such as that reported by Abu-Jaudeh, may require a lateral rhinotomy for removal but in most instances, the extraction is accomplished by intranasal procedures. Some patients will not tolerate the travail incident to the manipulation required for extraction of the growth under local anesthesia and for these individuals and children a general anesthetic is indicated.

It is unlikely that any but the small concretions may be extracted in one piece because of the brittle nature of the calculus and the small area through which it must be removed. The plan usually followed is to break off the most anterior portions with a Knight forceps and push the remainder into the nasopharynx for recovery through the mouth. The final fragment is usually deeply embedded and it may be necessary to grasp the stone firmly and rock it back and forth as a dentist loosens a tooth for extraction.

Removal of the rhinolith is not always a simple procedure and several sessions may be required before the mass is completely eliminated. This becomes apparent after the visible portions of the rhinolith have been removed and the operation becomes a groping procedure with a large segment of the calculus still unaccounted for. Bleeding is usually the factor which terminates each session. The usual bleeding attending intranasal procedures is intensified by trauma to the highly vascular granulation tissue in which the stone is embedded. In the event that the procedure is temporarily abandoned, a further attempt at removal may be undertaken a week later and in the meantime x-ray studies should be made in order to reveal the nature and exact location of any remaining fragments. Again, after removal of all visible and palpable portions of the calculus, x-ray studies are made as an assurance that no fragments have been overlooked.

Two cases of rhinolithiasis have been seen by us in the past twelve months. They have in common the fact that both appeared in women, were of several years' duration and were complicated by maxillary sinusitis on the involved side.

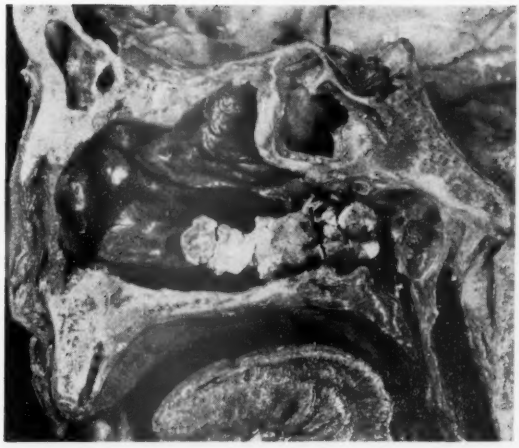

Fig. 1.—Rhinolith as it appears in specimen from cadaver.

## REPORT OF CASES

CASE 1.—Mrs. M. M., a 34 year old housewife was first seen on April 19, 1951, suffering from a nasal obstruction on the right side with a malodorous mucopurulent discharge, and pain and tenderness over the right cheek. The patient stated that her symptoms began four years previously, at which time she was treated by her family physician who later referred her to an otolaryngologist. Following x-ray examination she received treatment with nasal packs every week for four months. When this proved ineffective she was put on intensive penicillin therapy for ten days by her family physician. Subsequently she saw in order, another otolaryngologist, a chiropractor, who gave her cervical spinal adjustments covering a period of a year, an osteopath who carried out digital manipulations in the nasopharynx and finally, in desperation, a psychiatrist who referred her to us.

On examination the nose revealed a deviation of the external pyramid to the left with a septal deformity to the right which almost completely occluded that cavity. On posterior rhinoscopy there appeared to be a globular clump of glistening mucoid exudate filling the right choana. Transillumination revealed a dark right antrum.

Irrigation of the sinus produced a large quantity of foul smelling exudate containing strands and clumps of gray, putty-like material. Antral lavage was repeated a week later with a similar result. Following irrigation a nasopharyngoscope was passed into the right naris below the deviation of the septum. Progress of the instrument was stopped almost immediately as the tip scraped against a metallic-like object on the floor of the nose. Following application of local anesthesia the mass was grasped with a Knight forceps and a portion broken off. A few more bits of the rhinolith were obtained at this time, but the procedure was discontinued becase of excessive bleeding. On the following visit several more pieces of the calculus were removed and the remaining portion was found to be moveable and could be rocked in situ. This final piece was not removed at this time but a few days later it became dislodged and was coughed out by the patient.

Subsequent examination of the nasal cavity revealed the space to be lined with normal appearing mucosa, free of pus and foreign bodies. This was verified by x-ray examination which also revealed a clear antrum.

On gross examination the calculus was brownish gray in color, irregular in contour with blackened margins. Reconstructed it was 45 mm long (the largest intact piece being 35 mm) 12 mm in its widest portion and 18 mm in its greatest vertical dimension. It displaced 3.5 cc and weighed 4.35 gm. In the center of the largest fragment was found the probable nucleus of the rhinolith, a portion of a rubber band. Microscopic study of the putty-like exudate, recovered from the antrum on irrigation, was reported as amorphous, homogeneous material characteristic of caseous degeneration, thus indicating the presence of rhinitis caseosa concomitant with rhinolithiasis.

Case 2.—Mrs. M. P., age 72, presented herself on March 26, 1952, for relief from recurrent pain in the left cheek associated with a foul smelling purulent nasal discharge and blocked nasal cavity. This condition had existed for 14 years during which time the patient had been examined repeatedly by different physicians, mostly general practitioners. She was told there was a growth present which appared to be a polyp but she refused to submit to operative procedures for its removal.

Anterior rhinoscopy revealed a grayish, globular mass wedged in the left middle meatus between the lateral nasal wall and the sep-

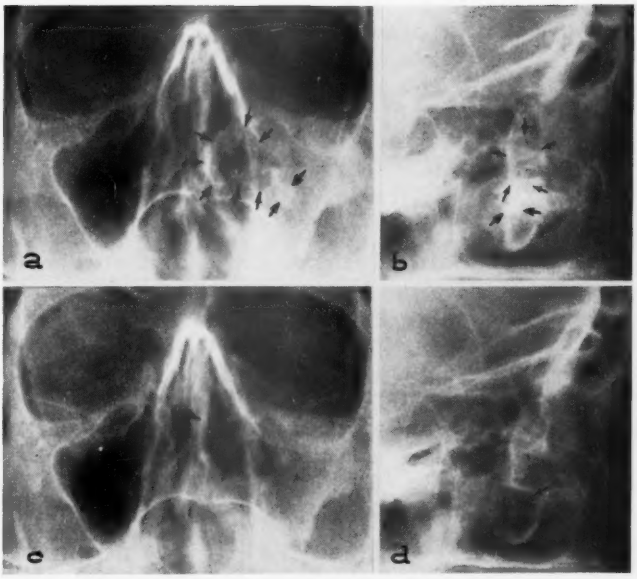


Fig. 2, Case 2.—A and B, Frontal and lateral views showing rhinolith in left nasal cavity. C & D, Same views, after removal of rhinolith.

tum. X-ray studies disclosed the presence of a slightly opaque mass in the left nasal cavity and a somewhat more dense object which appeared to be in the left maxillary sinus. When these were pointed out to the patient she consented to their removal.

Under local anesthesia the rhinolith was grasped with a Knight forceps. The mass disintegrated readily and seemed shell-like in structure. Considerable bleeding resulted and as the patient blew her nose minute particles of calcareous material were collected. X-ray films following this procedure showed absence of the cyst-like mass but continued presence of the small opaque object. On her next visit, a few days later, further manipulation in the nasal cavity dislodged this body and forced it into the nasopharynx to be hawked out by the patient. Irrigation of the antrum produced a thin, yellowish exudate extremely malodorous. Final x-ray films showed

SUMMARY

complete absence of the opacities previously visualized, although the left antrum appeared to be relatively hazy.

On a subsequent visit the patient related that the foul odor had disappeared and for the first time in many years she was able to breathe without a sense of obstruction. Antral irrigation at this time produced a clear return without odor.

Our reason for presenting this subject is to call attention to the fact that cases of rhinolithiasis exist and remain undiscovered for a number of years even though they present typical symptoms and are seen by many physicians.

Two cases were seen by us in the last twelve months. The concretions recovered in each case were not large as rhinoliths go, but nevertheless imortant to the patient's health. Both appeared in women, were of several year's duration and were complicated by maxillary sinusitis on the involved side. In one the sinusitis was of the caseous type and the rhinolith was formed around a foreign body which had become lodged in the nose, while in the other, there was a simple purulent sinusitis and no apparent foreign body formed the nucleus of the calculus. In both cases, as is common in most, the symptoms disappeared immediately upon removal of the foreign body.

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XLV

# THE USE OF CORTISONE AND CORTICOTROPIN IN THE FIELD OF OTORHINOLOGY AND LARYNGOLOGY

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The first rush of enthusiasm for the use of cortisone and corticotropin (ACTH) resulted in their indiscriminate and haphazard use in medicine. This enthusiasm has now abated to a large extent. We have been allowed time to assess the results of therapy with these substances and found them generally disappointing in the field of otorhinology and laryngology.

Conditions which seem to derive temporary benefit from the use of these substances appear to be those in which allergy appears to be an etiologic factor, especially if allergy is defined as being a localized type of autonomic dysfunction in which an antigen-antibody mechanism may or may not be present.[1]

## UNFAVORABLE EFFECTS OF TREATMENT WITH CORTISONE AND CORTICOTROPIN

For the purposes of this presentation it seems to me that a sounder basis for the use of cortisone and corticotropin will be established if the unfavorable effects of these substances are considered first and then the favorable effects on otorhinologic conditions.

*Euphoria.*—Sprague and co-workers[2] mentioned the frequent occurrence of euphoria especially during the early stage of treatment with cortisone. This may be a distressing complication, sometimes amounting to an acute mania. It is also possible that in some instances the good results attributed to treatment of conditions of the ear, nose or throat with these substances may depend to a considerable degree on the production of euphoria.

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*Effect on Carbohydrate Metabolism.*—Diabetes may be greatly aggravated by corticotropin or cortisone. Sprague and co-workers[2] stated that it is apparent that neither cortisone nor corticotropin in the doses which are usually employed produces any conspicuous alterations in the tolerance of carbohydrate. It is equally apparent, they said, that more striking changes in carbohydrate tolerance might be expected if the hormone was administered to persons having latent or frank diabetes.

*Effects on Appearance.*—The same authors[2] also stated that rounding of the facial contours or a Cushing-like appearance was one of the earliest signs of hormonal excess. Pigmentation of the skin also occasionally occurs after prolonged treatment with corticotropin.

*Disturbances in Water and Electrolyte Metabolism.*—Thorn and co-workers[3] found that retention of sodium chloride and water occurs with great frequency during treatment with corticotropin, but it rarely complicates treatment with cortisone.

Clinically, a progressive gain in weight is observed which presages frank edema. It is wise, therefore, to restrict the intake of sodium chloride and fluids of patients receiving corticotropin.

Thorn and his associates also found that a hypokalemic hypochloremic alkalosis might occur after treatment with either corticotropin or cortisone. This condition might be independent of retention of sodium chloride and water. It is associated with easy fatigability, muscular weakness and characteristic electrocardiographic changes. Oral administration of 1 or 2 gm of potassium chloride in enteric-coated tablets three times daily often will prevent and certainly will ameliorate these changes.

In passing, it is of interest that Thorn and his associates also found that abnormal elevations in blood pressure are seen only infrequently in the course of treatment with corticotropin and practically never in the course of treatment with cortisone.

*The Danger of Infection During Prolonged Treatment With Cortisone or Corticotropin.*—Both corticotropin and cortisone will suppress the resistance mechanism by which the body meets invasion from without by pathogenic bacteria and also the usual signs and symptoms of the infection.[4] As a consequence, patients who are

being treated with these agents may have a serious infection, especially septicemia, without showing the signs and symptoms by which such infections are ordinarily recognized. The physician must be on the alert for the development of such a complication.

*The Danger of Surgical Procedures on Patients Who Have Been Receiving Corticotropin and Cortisone.*—Sprague and his associates[2] presented evidence which suggested that cortisone acetate administered in ordinary therapeutic dosage would cause a depression of adrenal cortical activity. It is recognized, therefore, that the response of patients who have been recently receiving cortisone to the stress of surgical procedures resembles that of patients with Addison's disease. Many patients who have been receiving cortisone recently neglect to mention this fact to the surgeon. Because the period of recovery of the adrenal cortex is variable and may take from three to six weeks or more, it has become important that patients be queried specifically on this point before surgical procedures are undertaken. Patients who recently have received cortisone or corticotropin must be treated preliminary to and after operation in much the same way that a patient who has Addison's disease is treated.

## OTORHINOLOGIC CONDITIONS WHICH APPEAR TO BE FAVORABLY AFFECTED BY CORTICOTROPIN OR CORTISONE

Otorhinologic conditions which it has been suggested might be favorably influenced by corticotropin or cortisone include vasomotor rhinitis with or without polyposis, angioneurotic edema and idiopathic (lethal) granuloma of the midline facial tissues.

*Vasomotor Rhinitis and Its Sequelae.*—Carryer, Koelsche and associates[5] observed the nasal mucosa of patients with vasomotor rhinitis associated with hay fever and asthma during treatment with cortisone. The secretions tended to disappear first, then the color of the mucosa changed toward normal, and after a delay, the edema of the nasal mucosa receded. The following year Koelsche and associates[6] observed a less marked effect from further treatment of many of the same patients with cortisone.

Rose and associates[7] observed 8 patients with vasomotor rhinitis and polyps treated with corticotropin or cortisone. Six of these were improved temporarily, and in all 6 shrinkage of the mucous membranes and usually of the polyps occurred. The polyps, however,

never entirely disappeared. A small residue remained even in the most favorable cases and within two months of the cessation of treatment the polyps had returned. Rose and his associates stated, however, that the feeling of well-being "associated with the sense of smell and easy nasal breathing" created a marked change in the outlook of these patients. It seems probable that the elevation of mood observed in these patients resulted at least as much from the euphoria produced by these medicaments as it did from improvement of nasal breathing.

Rose and associates[7] found that for nasal polyposis the average effective dose of corticotropin was 100 mg per day in divided doses for the first three days after which the dose could be reduced to 50 or 60 mg per day. For some patients 100 mg per day were required continuously to produce a clinical effect. The dose of cortisone equivalent to 100 mg of corticotropin was found to be 200 mg per day.

Rose and his associates[7] observed that a second course of treatment with corticotropin or cortisone might not be as effective as the first, even though doses were the same and both courses were administered in the hospital. The reasons for this were obscure, but these observers suggested that a change in the time of the year or climate might have been a factor. Rose and his associates also noted that the condition of a number of patients appeared to be resistant to the use of both corticotropin and cortisone. These patients may have responded well to a first course of corticotropin, somewhat less effectively to a second course and not at all to a third or may have failed to respond to any but the first course. The same held true for cortisone.

Lake and Williams[8] treated 5 patients with severe polyposis with a total of 200 mg of cortisone a day given by mouth in divided doses, every six hours night and day. They found that 2 of the 5 patients failed to show any response as far as recession of the polyps was concerned even when at the end of three weeks the total dose was increased to 400 mg per day and treatment was continued for another week.. Three of the 5 patients had rather marked recession of polyps when they were receiving 200 mg of cortisone per day, in divided doses. The polyps of all 3, however, were as large as before treatment a month after treatment was stopped. In 2 of these patients treatment was repeated without any diminution in response. In 1 of these patients an attempt was made to delay the return of polypoid tissue by a maintenance dose of 50 mg of cortisone per day,

25 mg at night and in the morning. This treatment delayed the return of polyps slightly but had relatively little effect.

Dill and Bolstad[9] reported the treatment of 25 patients with allergic rhinitis with a spray of cortisone acetate (cortone acetate) 1 to 4 in saline solution. Each side of the nose was sprayed four times a day with this solution. Of the 25 patients treated, 12, or 48 per cent, obtained marked relief of symptoms. Six of the patients had some improvement and 7 none. In a second paper Dill and Bolstad[10] stated that of the 12 patients obtaining good results 5 continued under treatment and have been under observation for more than a year. In addition 10 new patients were treated. Of the entire 35 patients 14 had polyps on initial examination. The effect of cortisone spray was considered negligible in this group except that the polyps of 1 patient disappeared. The assessment of improvement in this group seemed to be based solely on subjective improvement such as return of the sense of smell and relief of nasal stuffiness. When relief was obtained, it lasted only as long as the spray was continued.

Lake and Williams[8] attempted to reduce nasal polyposis by the injection of 0.2 cc of cortisone acetate (cortone acetate) solution into all visible polyps daily. This treatment was continued for two weeks without an observable effect on the appearance of the nasal polyps.

Comment.—Perennial vasomotor rhinitis is a persistent allergic reaction affecting to a large extent the collagen of the tunica propria of the nasal mucosa. Nasal polyposis is essentially a collagen disease. Nasal polyps may produce more or less obstruction to nasal breathing. As a result of interference to drainage and the effect of allergic edema in lowering local resistance to bacterial invasion, infection of the sinuses may take place, producing a condition known as "hyperplastic sinusitis." Adenomyxomatous polyps or cysts in their early stages may be reversible phenomena. After long standing, however, they become fibrosed and will not recede even though the stimulus to allergic response is completely removed.

Heretofore nasal polyps and hyperplastic sinusitis have responded best when a combination of surgical, medical and specific anti-allergic treatment was given. Although these measures have not been entirely successful, their effectiveness in producing prolonged symptomatic relief is far greater than that of cortisone or corticotropin.

*Idiopathic (Lethal) Granuloma of the Midline Facial Tissue.*—Idiopathic granulomas of the midline facial tissues are being reported with increasing frequency. It seems probable that previously many of these cases were not reported, either because of the difficulty in making a definite diagnosis or because in such a long, lingering illness the patient frequently goes from physician to physician in hope of relief and consequently one physician is not often able to observe the total course of the illness. The use of cortisone and corticotropin for this group of patients seems to produce a healing tendency in these lesions.

Clinically the picture in idiopathic granuloma is one of progressive destruction of the nose, face and pharynx. The duration of the illness may be from a few months to five years or more. The most striking feature of the disease is the apparent complete absense of resistance on the part of the patient to the progress of the disease.

Hoover[11] described the typical pathologic features of a lethal granuloma of the face and nose. He stated that the center of the lesion was an amorphous necrotic mass. Peripheral to this there was an infiltration by inflammatory cells chiefly lymphocytes and macrophages with occasional plasma cells. A few polymorphonuclear leukocytes were also present. The large vessels in the tissue showed a definite perivascular cuff of lymphocytes and macrophages with degeneration of arterial walls in some regions. While there were focal accumulations of infiltrating cells in the tissues, the lesion was not characteristic of tuberculosis, syphilis or leprosy. To Hoover the histologic picture of the lesion was suggestive of that of the Arthus phenomenon.

Weinberg[12] in discussing idiopathic granulomas added what appears to be an important point. He found that these lesions were not strictly limited to the midline tissues of the face but that here and there throughout the body a pathologic picture of periarteritis nodosa developed. Weinberg stated that if all organisms which might produce a similar histologic picture were eliminated in so far as possible, there is left a picture which clinically consists of ulcerations of the upper respiratory structures associated with evidence of focal alterations in the lungs which pathologically consist of multiple granulomas of unknown origin. These may involve one or more organs with terminal development of periarteritis nodosa. He concluded that the constancy of the history of antecedent chronic infection in

these cases lends support to the belief that hypersensitivity may be a factor in the development of the lesion. From the viewpoint that allergy is fundamentally a localized hyperimmune reaction of the peripheral vascular bed, it seems reasonable to suppose that cortisone and corticotropin would tend to ameliorate this disorder. Williams and Hochfilzer[13] reported a preliminary trial of cortisone on a patient with a lesion which seemed to be of this nature. The effect on the lesion seemed to be encouraging although clinical trial was too short to be conclusive.

Moore and associates[14] reported the use of cortisone and corticotropin for a patient who had a relatively early lesion of this type with necrotic lesions in the nose, pharynx and larynx. Later in the course symptoms developed which suggested the presence of periarteritis nodosa. Under treatment with corticotropin and cortisone the lesions in the nose, pharynx and larynx healed completely and the general symptoms also subsided. At present their patient appears to be in a complete remission. This is the first report in the literature in which a patient with idiopathic granuloma has made an apparent recovery.

The dosage schedule used for this patient appears to have been inadequate to produce the most rapid and effective result. If dosage is based on the findings of Shick and co-workers[15] in the treatment of periarteritis nodosa, an initial dose of 300 mg of cortisone followed by 100 mg per twenty-four hours in divided doses given every six hours and continued for a minimal period of six weeks might be expected to produce a more rapid recession of the symptoms and signs.

## COMMENT

The use of cortisone and corticotropin (ACTH) in diseases of the ears, nose and throat seems to be as yet in the stage of clinical evaluation. With the exception of idopathic (lethal) granuloma, the conditions can be treated with reasonable success in most instances by other medical or surgical measures. For idiopathic granuloma, however, these hormones offer the only hope of saving the patient's life. It might be well, therefore, if the otorhinologist would delay a little in the indiscriminate use of these substances.

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XLVI

# BRONCHOGRAPHY IN CHILDREN

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Bronchography in children can be performed easily and safely. At the Hospital for Sick Children Toronto it has been carried out for many years; latterly with complete satisfaction to the paediatric and surgical services of this hospital.

However in the Annals of Otology, Rhinology and Laryngology September 1951,[1] an article stated that the technique of bronchography in young children is particularly difficult and the results poor. This may be the situation in certain hospitals but it does not need to be. This work is being done regularly by six members of the otolaryngological staff and is being taught satisfactorily to each resident in otolaryngology. There are thousands of bronchograms available for inspection.

The first report on our work[2] was made in the Journal of the American Medical Association of December 12th, 1942. The technique was modified and a second report[3] published in the Annals of O. R. & L. in June 1947. Both these papers discussed technique and results obtained in the old hospital in a room and under conditions which were far from ideal. The objections must have seemed so formidable to a casual observer that he may have found it difficult to appreciate that the work had been carried on as stated over many years with increasing satisfaction without any trouble of a serious nature.

In February 1951 we moved to our present new building where relatively ideal conditions for the carrying on of our work have been set up.

The pediatric staff of this hospital and the surgical staff are enthusiastic about every part of our procedure and it is because we believe that everything can be copied safely and readily in hospitals for children throughout the country that this subject is again being reported before this Society.

As stated in the other papers this work in our hospital commenced in 1927 and ever since has steadily increased. There is a gradual increase in the number of lobectomies. At present we perform bronchography regularly on two mornings a week, three or four bronchographies being performed at a session. The work is the combined and closely co-ordinated efforts of the staffs of otolaryngology, anaesthesia and radiology. The details of the technique employed have been slightly altered from time to time but the essential bronchoscopic method has been used since 1930. Because a new multiplane fluoroscope was provided in February 1951 certain modifications in the former technique became necessary and these will be described.

Bronchography by endo-bronchial injections of iodized oil through the bronchoscope under general anaesthesia and under fluoroscopic observation has been performed approximately 2,500 times. As from 3 to 5 bronchograms are made at each session we have approximately 10,000 films for inspection. The resultant bronchograms have led to about 111 lobectomies and pneumonectomies for bronchiectasis in the period since 1945 alone.

There have been no serious post-anaesthetic complications since the early years of our work. These were reported in the first paper from this hospital.

The object of the bronchography has been to provide accurate anatomical diagnosis of all portions of the bronchial tree. This is done primarily at the request of and for the information of the paediatric service. Where the abnormalities discovered are deemed suitable for surgical treatment the medical service enlists the interest of the surgical service. The bronchograms are studied in concert, and the requisite surgery performed.

In adults bronchography is performed adequately by the catheter technique. Occasionally this catheter technique can be used in suitably conditioned co-operative children who are relatively well. It is impractical to use it with miserable, coughing, sick, apprehensive, unconditioned children. It cannot be used at all in tiny children. On the other hand the technique at present in use in this hospital has made bronchography possible even in an infant one month old.

The conquest of bronchiectasis by the surgeon has made bronchography of great importance. Bronchography in children in this

hospital is of major importance to our surgical service. Failure to map a small portion of a lung has occasionally occurred as will be commented upon later in the paper but these occasions have been exceedingly rare.

Modern chest surgery can cure bronchiectasis. The thoracic surgeon excises every diseased portion of the bronchiectatic lung. Before he commences the surgical treatment he must know from the bronchogram the condition of every part of the lung. In the early days adequate mapping of the lingula was rare, of the right middle lobe uncertain and of the two upper lobes far from satisfactory. Our present technique enables us always to get the formerly difficult portions of the lung completely and adequately pictured. Our surgical service knows that at operation the lingula may seem normal by inspection and by palpation yet they have such confidence in our lung mapping that they unhesitatingly remove the lingula where our procedure has shown it to be diseased. The bronchograms which 5 years ago we might have considered good are no longer considered adequate. The chest surgeon knows that he cannot see all branches of the bronchial tree completely in any one picture. He requires exposures in certain positions for the outlining of special bronchi and exposures in other positions for the visualization of other branches.

Therefore, today, before he will operate upon a bronchiectatic patient, a chest surgeon requires for study bronchograms of all lobes of each lung in several positions. If he is not satisfied with the delineation of some part of the bronchial tree he will demand more bronchograms of that particular portion, for he must plan his surgical attack and he cannot do that until the bronchography is complete to the last segmental bronchus.

*Technique.* The preparation of the patient for bronchography varies with the age and condition of the patient. In some patients postural drainage is carried out for a period.

Bronchography in our hospital is performed in the bronchoscopic theatre of the department of otolaryngology. It is performed through the bronchoscope under general anaesthesia. The bronchoscopist is in charge of the whole procedure. But the operation is performed by a three man team, the anaesthetist, the bronchoscopist, and the radiologist. Each member in turn plays a major role. When the other members are playing their main parts the bronchoscopist

either co-operates with them by not interfering or by obeying their orders.

As the bronchoscopist is in charge of the procedure he observes the condition of his patient before the anaesthetic is started.

The bronchography is not preceded by sedation. Any sedative will interfere with the cough reflex if the dose is sufficient to diminish fear. The success of the procedure depends, in part, on the rapid return of the cough reflex once the anaesthetic has been stopped. No local anaesthetic is used.

The excitement of coming for operation excites coughing. The patient is turned on his side and the coughing is encouraged in order to free the bronchial tree of as much of its purulent material as possible. The anaesthetist attempts by gentleness and reassurance to gain the child's confidence. When he has achieved this he commences the anaesthetic.

The bronchoscopic operating room is 18 feet x 16 feet and has a high ceiling. There are many windows and a large door. Fresh air is delivered at the ceiling and the stale air is removed by suction through a ventilator, close to the floor and close to the head of the operating table.

The incoming air passes through filters. It enters at a rate which allows of complete change of air 7 times an hour. The rate is regularly tested once a month just before the regular monthly cleaning of the filters.

Relative humidity of the incoming air is 70%. The door of the operating room opens into a small corridor which is closed by doors from the main corridor. Keeping the windows and doors of the operating room closed for one hour before operation is necessary for maintenance of the humidity. The humidity is not constant but varies with the temperature of the room.

The result of this ventilation is that no concentration of explosive anaesthetic occurs and even if electrical sparks were produced they would do no harm.

However we take particular precautions to see that electrical sparks shall not occur. The table used is shock proof and completely grounded. The use of wool in the draping of the patient is prohibited. The view boxes and all the electrical outlets are flash proof.

The anaesthetic used is ethly-chloride-ether sequence. The child is placed in the lateral position with the face turned slightly down towards the table to permit saliva and discharges to flow out of the cheek rather than to collect in the pharynx. This position makes maintenance of the airway easier as anaesthesia progresses and relaxation occurs. Suction is available at all times and is used as necessary. The induction stage is not hurried: it being our belief that some coughing at this stage is helpful in clearing secretions from the airway.

The open mask method is used until sufficient depth is obtained to allow the introduction of the bronchoscope without damage to the patient or embarrassment to the bronchoscopist. The mouth and pharynx are then cleared with a suction tube and the patient turned on his back for the exposure of the larynx.

A bronchoscope suitable to the size of the patient is now introduced and from this point anaesthesia is maintained through the bronchoscope by means of a rubber tube delivering oxygen and ether vapor produced by the AINMED gas anaesthetic apparatus.

At this point the depth of the anaesthesia is diminished to allow slight coughing to occur. The purpose of this is to promote the expulsion of secretions from the inaccessible distal portions of the bronchial tree to the main bronchi from which the secretions can be sucked by the bronchoscopist. The attempt is made to clear the bronchial tree as far as possible. Iodized oil will not enter a plugged bronchus. Much of the success of the whole procedure depends upon this evacuation of secretions from the main bronchi by suction. (The material aspirated is collected and sent to the laboratory for microscopic and bacteriological investigation.) When there are no more bronchial secretions to be removed by suction the bronchoscopist notifies the anaesthetist who then proceeds to increase the anaesthesia. Deep anaesthesia has to be reached and maintained for about five minutes. The anaesthetist knows from experience, when his patient is ready for the next stage.

The next stage is the introduction of cool iodized oil into the bronchial tree to be outlined. This is done through the bronchoscope via the metal cannula.

At this point the radiologist is summoned. (The radiological department immediately adjoins the bronchoscopy room.)

For bronchography of the right lung, assistants turn the patient's body to the left anterior oblique chest position and use sand bags to maintain the position.

(The patient's body is rotated 45% to the right. The left shoulder is uppermost and therefore close to the film which is in the carrier above it. In this position the right main bronchus and its branches are dependant, show up individually and are in the best position for filling. Our radiologist has found that no particular advantage is gained when filling the bronchial tree by lowering the chest or elevating the feet.)

The oblique positions are desirable because the bronchial tree divides in a direction oblique to the horizontal (when the patient is in the horizontal position). In consequence the oblique position permits clear separation and identification of the secondary bronchial divisions. The iodized oil is delivered through a special metal tube into the mouth of which a modified Burrell's syringe is fitted and locked by the nurse. The room is now darkened and the radiologist using the fluoroscope directs the bronchoscopist until the bronchoscope and the oil tube are in the requisite position, just proximal to the opening of the bronchus to the right upper lobe. The beveled end of the bronchoscope is directed laterally. At this point the radiologist asks the nurse to inject the iodized oil slowly. He watches the upper lobe bronchi fill until they are well delineated. At his command the injection of the oil is stopped and at that moment a "spot" film is taken. This film is invariably excellent and without any flooding of alveoli.

The radiologist then directs the bronchoscopist to push the bronchoscope and the oil tube slightly further into the right main bronchus and directs the nurse to inject more iodized oil. This outlines the right middle lobe and the right lower lobe and when the optimum filling has occurred a second "spot" film is taken. At this moment the "spot" film will show a clear outline of the middle and lower lobes but the upper lobe will now commence to show flooding or alvcolarization.

Following this the patient is returned to the horizontal position and a postero-anterior "spot" is taken.

At this point it is our custom to pull out the oil cannula and pull up the bronchoscope so that its lower end is just above the carina.

Then the patient is turned to the left side into the right lateral position and a lateral "spot" is taken.

The oil is never forced in by the syringe—it is drawn into the smaller bronchi and from there into the bronchioles by inspiration. These distal parts fill only if sufficiently clear of secretions and if that part of the lung expands with inspiration.

The key note of good results lies in the absolute co-operation of bronchologist and anaesthetist and radiologist. All the pictures must be taken as rapidly as possible. Deep anaesthesia until the last exposure is essential.

At the conclusion some of the iodized oil is removed by suction and when the anaesthetist is satisfied with the patient's condition the bronchoscope is pulled out.

The patient comes rapidly out of the anaesthetic. Because there has been no pre-operative sedation the cough reflex returns rapidly and the iodized oil is expelled. It is extremely rare that there is any embarrassment to those watching the post-anaesthetic recovery. The work is performed in the morning between 10 and 12:30 and the child is usually sitting up by 4:30 p. m. and enjoys his supper and is discharged almost invariably the next morning.

Bronchography of the left lung is done on a subsequent occasion, after an interval of several weeks. The main consideration is that most of the iodized oil injected at the first bronchography should have vanished so that the delineation of the left bronchial segments is not impaired by overlying iodized oil in parts of the right lung.

For bronchography of the left lung the patient is first placed in the right anterior oblique position. After the filling of the left upper lobe great attention is paid to the proper mapping of the bronchus leading to the lingula. The successive positions of the patient are the exact reverse of those used in the mapping of the right lung.

Delineation of all parts of the lung is very satisfactorily accomplished by our present technique. Occasionally it happens that some segment of one lung will not expel the material filling its smaller bronchi and consequently it cannot be filled with iodized oil. Occasionally injection of iodized oil is followed by more adequate emptying of the bronchial tree and a second bronchography some weeks

later is successful in outlining the segment which formerly would not fill. There have been occasions when after lobectomy the unmapped segment of the lung has been found almost completely filled with semi-solid material which could not possibly have been expelled.

In the two papers already published modifiications of the above technique have been mentioned. All of these have now been discarded.

In the literature bronchography in young children is described as accompanied by certain theoretical dangers. Four of these warrant comment. The first is the risk of the anaesthesia. We have described our methods of procedure under general anaesthesia. We have at the hospital a staff of ten certified anaesthetists and in their hands we do not believe that we now have any risk whatsoever. In our long experience we have been asked to bronchograph many very young children, many with extreme degrees of bronchiectasis, and other patients with grave conditions for carefully considered medical reasons. We do not approach such work haphazardly but on the contrary with great caution and always with every step mutually discussed by the three co-operating services. We can say without reservation that what we can do and have done at our hospital can be done elsewhere with equal satisfaction, if similar co-operation is obtainable.

The second risk put forward is that of the technical procedure of the introduction of the iodized oil. We use as stated a bronchoscope. We use always a bronchoscope relatively small for the child's larynx. We do not know of a single instance in which trauma has resulted. As already stated we teach this procedure to our residents and have done so without any damage to our little patients. We do not believe at all the statement that—"it seems nevertheless advisable to resort to bronchoscopy only when other techniques cannot be applied or are inadequate." Between us we have inspected techniques in vogue in other hospitals. In a few instances we have seen a technique neatly and dexterously performed with good bronchography but we have not seen in any other hospital bronchography performed by such a variety and such a large group of people nor in anything like the volume performed at our hospital. The results that we have seen elsewhere have been occasionally good but we can maintain without exception, that all our cases are essentially good and the great majority are excellent at the first trial.

In our hands, the third danger mentioned, the risk of respiratory trouble due to the filling of the tracheo-bronchial tree has never occurred. Our filling is done under the direct vision of the radiologist using the fluoroscope, consequently excessive filling is not allowed to occur.

The fourth danger, the risk of intoxication due to iodine we have never seen.

Certain other objections are to be found in the literature. One of these was that general anesthesia slows down the spontaneous expulsion of the oil suspension through coughing and expectoration. This statement is simply untrue. Ether anaesthesia stimulates spontaneous expulsion of the oil suspension through coughing and expectoration. As described above we use this ether-aroused expulsion to evacuate bronchial secretions and then we, for a very short time, have the anaesthetic deepened so that the iodized oil may remain in the distal bronchioles for a long enough time to enable the radiography to take place. Anaesthesia beyond this point is unnecessary and is not carried on. The termination of the anaesthesia permits expulsion to be rapid and in most cases the bronchial tree is quickly cleared.

In certain hospitals a notice can be found posted in the bronchoscopy room to the effect that no general anaesthetic is to be given when the radiological unit is to be used. And yet in the same hospital in a room of similar size exactly the same radiological apparatus is used many times daily while patients under general anaesthesia are having work performed by surgeons treating fractures and dislocations.

I do not want to be misunderstood. We recognize dangers of all sorts and we never neglect these dangers. We take every precaution that we know of to avoid trouble.

There is always danger to the patient when an instrument carrying an electric bulb is inserted into a patient's pharynx or bronchial tree when the patient is under anaesthesia. This danger is present every day in every hospital in the land where anaesthetists putting a patient under a general anaesthetic use a laryngoscope for the insertion of the endotracheal catheter. There is always danger from a possible electric spark when the patient is being given an anaesthetic of an explosive nature.

The way to avoid trouble is to avoid the use of the more explosive mixtures when a laryngoscope or a bronchoscope has to be used, and always to make sure before inserting it that the electrical attachments are in perfect working order.

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XLVII

# THE INFLUENCE OF THE CHEMOTHERAPEUTIC AND ANTIBIOTIC DRUGS ON THE INCIDENCE AND COURSE OF DEEP NECK INFECTIONS

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That the antibiotics have remarkably influenced the incidence and course of bacterial infections in all parts of the body is universally recognized. The object of this paper is to evaluate their influence on infections in and near the neck. Such infections in the past, while not occurring in great numbers, were not uncommon.

For purposes of comparison two groups of cases will be used, namely, one group of 78 cases reported in 1939 before chemotherapy,[1] and a group which consists of cases observed since 1940, during which time the sulfonamides and, later, the antibiotics were used.

The comparison demonstrates a recognizable change in the incidence of the entrance portals and the regions of involvement.

In the first group of 78 cases seen before 1940 there were 56 cases in which the portal entrance was the pharynx, tonsils and adenoid . In the second group of 55 cases the infection entered through the pharynx, tonsil and adenoid region in 18 cases. The percentages are 71 and 32 respectively.

There is also a striking change in the relative and total incidence of the dental group. In group 1 there were 16 cases of dental origin, 20 per cent, as compared with 19 cases, 34 per cent, in group 2. Another significant observation may be made, namely, the increased frequency of salivary glands as a portal of entrance. There were no salivary gland cases in group 1, while in group 2 there were 11 cases, nine in the submaxillary and two in the parotid. This is a fairly large percentage, 20 per cent. The only explanation that occurs to me is that at that time I had not fully realized that salivary gland infections are actually infections of the neck from the start. What

TABLE I.—PORTALS OF ENTRANCE.

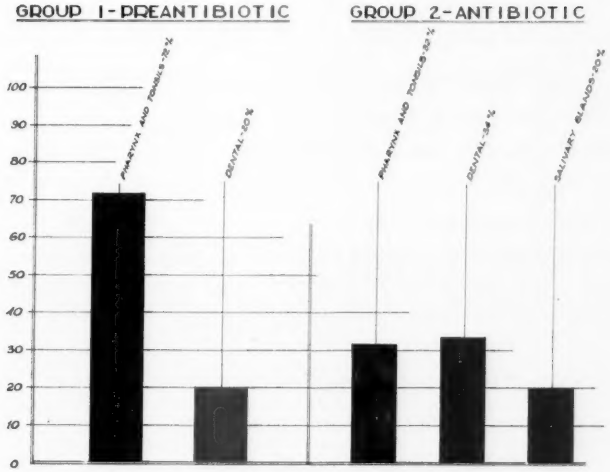
| | | |
|---|---|---|
| **Groupe 1.—78 Cases.** | | |
| Pharynx and tonsils | 56 cases | 71% |
| Dental | 16 cases | 20% |
| Lingual tonsil | 1 case | |
| Petrous temporal bone | 1 case | |
| Gingival Trauma | 1 case | |
| Sialitis | none | |
| Not determined | 3 cases | |
| **Group 2.—55 Cases.** | | |
| Pharynx and tonsils | 18 cases | 32% |
| Dental | 19 cases | 34% |
| Submaxillary sialitis (9) | | |
| Parotid sialitis (2) | 11 cases | 20% |
| Lingual tonsil | 1 case | |
| Scalp furuncle | 1 case | |
| Not determined | 5 cases | |

few there may have been should have been included. Later, in 1942, I wrote a paper on salivary gland inflammations, and in that paper stated that they are in reality neck infections.[2]

Infections from the various other portals of entrance occurred in each series sporadically and the occasional appearances are so few as to make comparisons insignificant.

When the infection is seen while it is still mostly confined to the portal of entrance, and treated promptly with antibiotics, further extension is apt to be stopped and the infection soon eradicated. How many of these would have extended to the neck if they had received no antibiotics is impossible to estimate, but it may be reasonably assumed that the number would have been larger. The pediatrist and the general practitioner today treat cases which in former times

TABLE II.—PORTAL OF ENTRANCE.




were treated by the otologist and the rhinolaryngologist. A short period of treatment with the antibiotics terminates the infection at the portal of entrance, and there can be no doubt that many deep neck infections are being prevented. Occasionally there appears a patient who has a well developed infection because early medical advice was not sought. Also occasionally there appears one whose infection does not yield readily to antibiotic treatment, and this type will be discussed later. I have in the past stated that I believe that infections arising in soft parts seem to yield to antibiotic medication more readily and more rapidly than infections having their origin in bone tissues such as dental-alveolar and the temporal bone.[3]

Since there have been these significant changes in the incidence of the portals of entrance it may be appreciated that there will also be changes in the incidence of the regions of inovlvement. The same two periods of grouping are again used.

*Pharyngomaxillary Infection.* In group one pharyngomaxillary infection occurred 42 times or 53.+ per cent. In group two it occurred 16 times or 29.+ per cent. This indicates a very considerable

TABLE III.—REGIONS OF INVOLVEMENT

Group 1.—78 Cases.

| | | |
|---|---|---|
| Pharyngomaxillary (Parapharyngeal) | 42 times | 53% |
| Submaxillary (Ludwig's angina) | 18 times | 23% |
| Visceral (Buccopharyngeal and Pretracheal) | 8 times | 10% |
| Carotid Sheath | 11 times | 14% |
| Prevertebral (Retropharyngeal) | 3 times | |
| Lymphadenitis, with (18) and without (26) abscess | 44 times | 56% |

Group 2.—55 Cases.

| | | |
|---|---|---|
| Pharyngomaxillary (Parapharyngeal) | 16 times | 29% |
| Submaxillary (Ludwig's angina and Parotid) | 32 times | 58% |
| Visceral (Buccopharyngeal and Pretracheal) | 4 times | 5% |
| Carotid Sheath | 2 times | 3% |
| Prevertebral (Retropharyngeal) | 1 time | |
| Lymphadenitis, with (11) and without (13) abscess | 24 times | 43% |

reduction of pharyngomaxillary infections. It is my considered opinion that the antibiotics have brought about this reduction in the occurrence of pharyngomaxillary infection.

*Submaxillary Type Infection.* In group one the submaxillary type occurred 18 times, or 23.+ per cent In group two it occurred 32 times, or 58.+ per cent, the largest single type. It is thus disclosed that the most significant change is this predominance of the submaxillary type. It is my belief that one of the reasons for this is that infection from the dental region has not been reduced, or if so, not to the same extent as has the pharyngomaxillary type. The most probable explanation is that dental infection does not usually receive antibiotic medication until the infection is already beyond the dental areas in the neck; whereas pharyngeal, tonsillar and upper respiratory infections usually receive antibiotic medication while the infection is still limited or confined to the portal of entrance. Another reason is that sialitis, parotid or submaxillary, was the portal of en-

trance in 11 cases in group 2 resulting in submaxillary region localization. When infection originates in the salivary glands it is in the neck from the start. It is sometimes difficult to determine whether the infection is in the salivary gland itself or in the lymph nodes in relation with it. Usually when the infection is primarily in the salivary gland there will be sudden and very uncomfortable swelling when eating. It is also often possible to express pus from the duct by massaging the gland. Lemon-sucking may also aggravate the discomfort. None of these symptoms and signs are present with lymphadenitis alone.

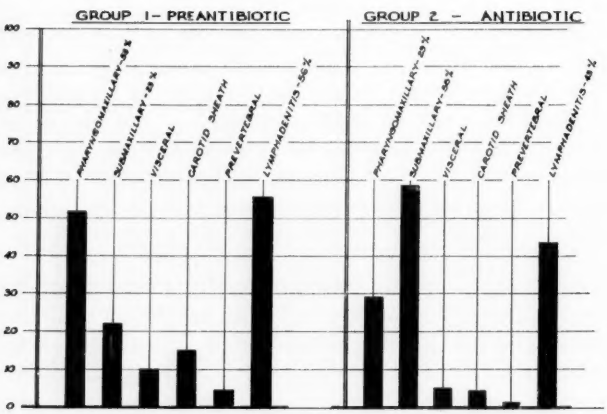
*Lymphadenitis.* It has been observed in a number of instances that lymphadenitis may localize and form an abscess while intensive antibiotic treatment is being administered. Fluctuation frequently develops over a period of a few days. This secondary localization in deep cervical nodes has been observed after an abscess in the parotid gland was drained.

Lymphadenitis, with and without abscess, was present 44 times, or 56 per cent, in group 1, 18 times with abscess, and 26 times without abscess. In group 2 it occurred 24 times, 11 times with abscess and 13 times without abscess. This discloses a lower percentage, 43 per cent, in group 2, and while no dogmatic conclusions may be drawn one may at least assume that despite antibiotics the lymphatic system is still very actively concerned, in a protective sense, in the presence of infection.

*Visceral Fascia Infection.* Infection along the visceral fascia occurred eight times in group 1, or 10 per cent, as compared with four times in group 2, or 5 per cent. It is reasonable to attribute this reduction to antibiotic therapy, not only when applied before the infection has extended into the neck and into the walls of the viscera, but also for the reason that more endoscopists are trained in skillful foreign body removal. Antibiotic therapy is very effecacious in preventing infection from minor traumas incident to foreign body removal, especially foreign bodies in the esophagus.

*Prevertebral Fascia Infection.* Prevertebral fascia infection, or retropharyngeal abscess, occurred three times in group 1, or 3.+ per cent, and once in group 2, or 1.+ per cent. Because of the small number of cases percentages here have no significance, and it may be concluded that no change has been brought about. Retropharyngeal

TABLE VI.—PURE CULTURES.




abscess occurs usually during the first two years of life, and therefor may be expected to remain about as formerly for the reason that the infection in infants is fully developed before it is recognized and before antibiotics have been administered.

*Carotid Sheath and Blood Stream Infection.* Carotid sheath infection with or without blood stream infection occurred 11 times in group 1 or 14 per cent, and twice in group 2, or 3 per cent. In one of these two cases the patient had a number of chills with excessive sweating, but the blood cultures were negative. She was treated with sulfonamides before penicillin was available and was the only death in group 2. She was extremely septic when first seen and the death was from septicemia.

This shows a significant reduction in blood stream infections in the antibiotic group, and is evidence that the antibiotics exert a pronounced protection against sepsis. This is markedly reflected in the appearance and general demeanor of the patient. Under intensive antibiotic therapy it is rarer for a patient to have chills. There is, however, some diaphoresis but much less in evidence than when no antibiotics were available. Therefor dehydration is also much less in evidence than formerly. Moderately high septic blood counts are present in the beginning, but after a few days of antibiotic therapy

the total white cell count and the neutrophile percentage are reduced. The non-segmented neutrophiles, or young forms, are not so much increased when the antibiotics are given. The shift is still there, but less.

In pre-antibiotic days the eosinophiles promptly disappeared from the blood count in the early intense stage of the infection, and their reappearance was interpreted as a sign that the infection was no longer so severe. When antibiotics are administered early in the infection the eosinophiles frequently do not disappear from the count, but remain throughout the course of the disease.

Sometimes, particularly when the sulfonamides are being given in full dosage, there will occur leucopenia and neutropenia, and, perhaps, agranulocytosis. In such cases the sulfonamides should be discontinued, but penicillin may be continued, and folic acid administered, an initial dose of 100 mg and thereafter 20 mg daily for adults. In these cases the temperature is apt to remain high until the folic acid is added to the therapy.[4, 5]

If the infection has been prolonged there may develop an anemia which requires appropriate medication, but in infection whose duration is not prolonged anemia is usually of mild degree only.

The absence of general symptoms and signs of sepsis makes it necessary for the clinician to rely more on the local manifestations at the site of the infection, and when these are obscure and not frank he may be faced with quite a dilemma in evaluating the course of events. The optimum time for drainage and the very indications for any drainage at all are thus much more difficult to determine. At such a time it will be necessary to penetrate or see through "the penicillin fog," an expression I first heard used by Dr. H. P. Mosher. Until the infection is really under full control and practically cured the sedimentation rate will remain high. In the presence of a blood count which is not much increased considerable diagnostic help is derived from the sedimentation rate. If this is normal the sepsis may be regarded as mild or absent.

Under penicillin medication the total white cell count is lowered sooner than the neutrophile percentage. This is also true after a drainage operation. Penicillin does not disturb the blood count as much as do the sulfonamides. With either there may be expected a

lowering of the leucocytosis and the neutrophile percentage, but with the sulfonamides this is apt to be excessive and is largely a toxic manifestation in that there occurs an abnormal leocopenia and agranulocytosis. This toxic effect is not often, if ever, caused by penicillin which lowers the total and neutrophile counts indirectly by removing the infection, and not by any toxic action on the hematopoietic organs.

### BACTERIOLOGY

The information which is derived from routine bacteriological investigation in these cases of neck infection is quite confusing, and the results as recorded on the chart often seem inconsistent. In those patients who have recovered without surgery the culture has not been taken from the inflamed tissues of the neck, but from the portal of entrance which may have been the pharynx, tonsils, dental region or mouth. It is therefor usually a mixture of organisms, any one of which may be the invader of the neck. The evidence is therefor highly inferential. Sometimes organisms from an abscess do not grow on cultures and a report "no growth" is received, and this even when a large quantity of pus from a large abscess is transferred to the culture medium. The organisms may have been rendered incapable of growing by the antibiotic therapy. However, it must also be remembered that such "no growth" reports were encountered before the antibiotics came into use. Also, pathogenic organisms may be cultured from a perfectly normal mouth and throat. A bizarre combination of organisms is often reported; in fact these combinations comprise a majority of the throat culture reports. After drainage we look for and hope to get a pure-culture report, for then we may be reasonably certain that we have adequately reached the infectious location. In this connection, however, it must be remembered that even though a pharyngomaxillary abscess has been drained high up, there may still occur an abscess of the deep lymph nodes lower down on the sheath.

Chemotherapy has, if anything, added to the difficulties by inhibiting the growth of organisms on culture. If there has been sufficient time bacterial sensitivity tests will sometimes help by indicating the selection of the most effective antibiotics. The tests should be started as soon as cultures are available. It has been customary to use the throat and mouth cultures for this purpose. Sensitivity tests are not mathematical certainties. I have seen infections yield to

TABLE V.—PURE CULTURES.

| | |
|---|---|
| Group 1.—Preantibiotic 78 Cases 39 Times 50% | |
| Streptococcus hemolyticus | 24 times |
| Streptococcus non-hemolyticus | 3 times |
| Streptococcus viridans | 2 times |
| Streptococcus, not typed | 3 times |
| Streptococcus hemolyticus in blood stream | 1 time |
| Staphylococcus aureus, not typed | 1 time |
| Staphylococcus albus, not typed | 3 times |
| Staphylococcus albus hemolyticus | 1 time |
| Pneumococcus | 1 time |
| Group 2.—Antibiotic 55 Cases 16 Times 29% | |
| Streptococcus hemolyticus | 3 times |
| Streptococcus non-hemolyticus | 2 times |
| Streptococcus viridans | 1 time |
| Staphylococcus aureus hemolyticus | 5 times |
| Staphylococcus aureus, not typed | 4 times |
| Staphylococcus albus, not typed | 1 time |

penicillin therapy when sensitivity tests were reported resistant to penicillin. When the neck abscess has been reached by surgical drainage recovery is usually so rapid and satisfactory that sensitivity tests will not be necessary.

A comparison of pure cultures in the two groups is noteworthy and somewhat striking. In group 1, 78 cases, pure cultures were found 39 times, or 50 per cent, of which 24 cases were streptococcus hemolyticus. In group 2, 55 cases, pure cultures were found 16 times, or 29 per cent, and the streptococcus hemolyticus occurred only three times. No growth was reported as often, four times, in the 55 cases of group 2 as in 78 cases of group 1.

Several conclusions seem justifiable: (a) there is not the predominance of the streptococcus hemolyticus in the antibiotic group that there is in the pre-antibiotic group: (b) the antibiotics do exert a definite inhibitory effect on the growth of organisms on culture; (c) the antibiotics, particularly penicillin, have had their best effect on the streptococcus hemolyticus; and (d) the other organisms now encountered, such as the staphylococci, are more resistant.

### CURES WITHOUT SURGICAL DRAINAGE

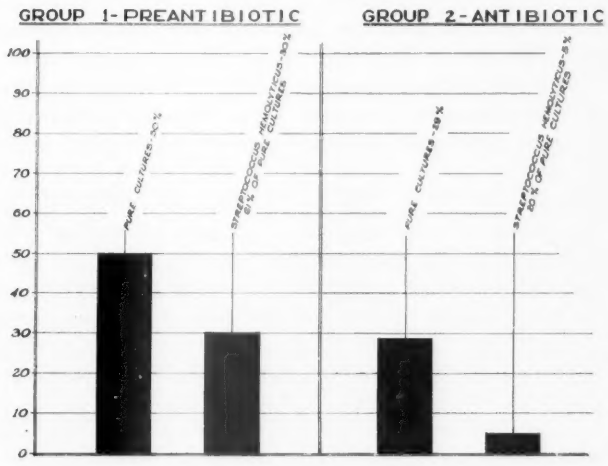
In the pre-antibiotic group 1 there were five cases in which there was subsidence of the infection without surgery, 6.+ per cent of the 78 cases. One case, not included in these, had an abscess which ruptured spontaneously through the tonsil fossa and recovered without surgery.

In the antibiotic group 2, seven cases were cured by the antibiotics without surgery. The longest stay in the hospital was ten days and the shortest, three days. There were five cases of the pharyngeal and parapharyngeal type in one of which the hypopharynx and extrinsic larynx were also involved. The remaining two were the submaxillary type, both of dental origin, one developing a severe well-marked Ludwig's angina which at first was fully expected to require surgical drainage. She was discharged cured on the tenth day without having had any surgery. Those seven cases comprise 12.+ per cent of the total 55. Therefor, it may be justifiably stated that between 10 and 15 per cent of neck infections may be terminated with antibiotic therapy if seen early.

Since writing this paper I have treated another case with suprahyoid dentoalveolar infection which recovered under penicillin therapy and without surgical drainage. There was extreme trismus, and a frank Ludwig's angina was already present. If surgery had become necessary a preliminary tracheotomy would have been unavoidable.

How often an incipient infection is aborted at the portal of entrance when therapy is administered at such a time it is impossible to estimate, but there must be a great number. Wanamaker et al.[5] reported on the effect of penicillin and aureomycin on a series of 475 cases of pharyngitis and tonsillitis. Results were better than without treatment. Suppurations were unusual in all groups. Seven cases later developed acute rheumatic fever, five in the untreated control group. Comparatively the results with penicillin and with

TABLE IV.—REGIONS OF INVOLVEMENT.




aureomycin were about the same, but penicillin prevented the carrier state while the aureomycin did not. To me it is surprising that there were not more cervical lymph node infections and complicating suppurations. They still occur.

## COMPLICATIONS

It will be more revealing to mention these in the absentee sense. In the 78 cases of group 1 there were 54 cases with no complications, or 69 per cent. When complications did occur they were severe and dangerous. In the 55 cases of group 2 there were 34 cases with no complications, or 61 per cent. Accordingly complications have not been reduced, but have changed in kind and degree. In group 2 there were two cases of leucopenia and two cases of toxic nephritis caused by the sulfonamide medication, and there were three cases requiring preliminary tracheotomy for restoration of the airway despite sulfonamide medication in one case and penicillin in the other two. The two which received penicillin were dyspneic early, before admission to the hospital. The sulfonamide case was in an advanced stage of Ludwig's angina, and tracheotomy was necessary before anesthesia could be administered. There was only one case with blood

stream infection in group 2. She had severe chills even before admission though blood culture remained negative. She did not yield to sulfonamides and drainage, and died on the fourth day after admission. The type of infection was Ludwig's angina, bilateral. This was the only death in the antibiotic group. One case had a strongly positive Wasserman reaction, and two had diabetes. Two others had had coronary thrombosis a short time prior to the neck infection.

The outstanding revelations pertaining to complications are:

1. The rarer occurrence of blood stream infection in the antibiotic group. In the pre-antibiotic group the internal jugular vein was ligated or resected in four cases and in none of the antibiotic group. There were 2 cases of arterial erosion in the antibiotic group, for which ligation of the common carotid was done in one case and ligation of the external carotid in the other.

2. Severe and permanent damage to vital organs such as the heart, lungs, liver and kidneys did not occur in the antibiotic group. The importance of this cannot be overestimated.

3. The necessity for tracheotomy has been increased because of the greater incidence of the Ludwig's angina type of infection.

## DIAGNOSTIC DIFFICULTIES

Formerly the local and general manifestations were frank and severe. The patient was very uncomfortable locally and very ill generally. The antibiotics change such a picture very soon after administration is begun. The local manifestations are much less uncomfortable and the general manifestations are so much ameliorated that the patient seems not nearly so ill. Frank blood stream infection is now a rarity, and while there may some diaphoresis, it may be very easily overlooked if no inquiries are specifically made. Digestion is not so much deranged; chills are not so commonly present. For all these reasons dehydration is not the pronounced problem it formerly was. The changes in the blood count are not so pronounced, at least not after antibiotic medication has been well established, for which reason lower blood count readings must receive more consideration than similar counts would have received formerly. The sedimentation rate should be more frequently done than formerly for it may continue to be increased when the count is hovering around the upper normal levels.

If an abscess deeply situated is already in existence progressive enlargement or extension may be checked, and a more or less static condition established. In other words it will remain as a small abscess which is difficult to detect. Surrounding tissue inflammation and edema may show marked subsidence. If at such a time an abscess can be detected, or even strongly suspected, surgical drainage will be rapidly curative, and it is probable that a less extensive operation will be entirely adequate. If frank fluctuation is present there will be no diagnostic problem, and surgical drainage is immediately indicated. When fluctuation is absent or doubtful, edematous pitting on finger pressure may be regarded as evidence of underlying pus. In cases in which the process is diffuse and cellulitic, and there is not yet any localization, antibiotics without surgical drainage are often curative. In the necrotic tissues of an abscess there is no blood circulation and therefor antibiotics do not reach such tissues. Both horns of the dilemma, local and general, must be rendered harmless by being capped or removed. Formerly in an extremely severe infection the imminence of blood stream infection justified surgical drainage long before any evidence of fluctuation was present. In many cases no free pus was encountered, and the operation prevented blood stream infection. If the disease does not in these days yield to antibiotic therapy in a reasonably short time, two to four days, surgical drainage is indicated as in preantibiotic times. When in doubt drainage is still the safest course. Diagnostic acumen is now in much greater demand.

### ANTIBIOTIC THERAPY

All of the cases in group 2 were treated with either the sulfonamides or penicillin, or both. Only in one or two cases were any of the others used, chiefly for the reason that until 1943 only the sulfonamides were available to us, and later, because the infections yielded so quickly and so conclusively to penicillin, with and without drainage, that there were no further indications for any kind of medication. No cases were encountered in which penicillin could not be administered. There were none that showed any of the allergic manifestations we now encounter more frequently. Because large numbers of patients have received penicillin for previous illnesses of many kinds a greater number of patients in the future will have developed a sensitivity to penicillin. The chief concern or hazard with penicillin therapy is the possible development of penicillin-

resistant organisms. During the course of any one illness this possibility is not common because the course is usually of short duration and organisms are not apt to become resistant in so short a time. In the event of resistance of organisms other antibiotics are now available. The same may be said with regard to allergic sensitivity, but penicillin is to be regarded as the most effective and least toxic of all the antibiotics now available. When a frank abscess is already existent, and prompt drainage is applied it is frequently unnecessary to give any antibiotic therapy. Antibiotic therapy alone, without drainage, does not cure such cases. If no abscess is reached by drainage, penicillin therapy is very important. Penicillin G is now the commonly used preparation. In cases of allergic sensitivity it will be necessary to look for substitutes. A newer preparation, penicillin O has been evolved, which is designed for such contingencies. Others are also becoming available. While penicillin administered orally has its advocates[7] who show proof of adequate blood levels, I have not used it, fearing that allergic sensitivity is more likely to be produced. It is now well known that penicillin lozenges frequently cause very marked stomatitis and gastrointestinal disturbances. In these extremely acute and fast-traveling neck infections hospitalization and intensive parenteral medication are prime necessities. If the patient has had no antibiotic medication it is well to start with one dose of penicillin G in saline by vein and at the same time one dose intramuscularly; then continue with an intramuscular dose of the same every 3 hours around the clock. This is continued until conditions show marked improvement, perhaps after drainage if that becomes necessary, or after the improvement indicates that drainage will not be necessary. After that one of the newer repository-type preparations may be substituted, one or twice daily to lessen the number of needle punctures and to avoid disturbing the patient during sleeping hours. The original aqueous penicillin G appeals to me greatly because of its rapid absorption and effects. The average dose of the aqueous solution is 100,000 units every three hours for adults, and that of the repository-type 300,000 or 400,000 units once or twice a day according to the severity of the illness. Until now I have not had to give penicillin O in neck infection cases.

During the war when penicillin was very scarce and strictly allocated, results seemed very striking even with the small doses of the crude preparations which were then available. At that time the eter every two to four hours. The strength of this solution is more

usual dose was 20,000 units for adults. Can it be that organisms have acquired increased resistance, and that larger doses are now necessary? Or have antibiotics which patients have repeatedly received rendered the natural immunizing mechanisms of the body less active? It could be partly both. It is now believed that the ordinary head cold is instigated by a virus infection which in practically all cases sets the stage for invasion by bacteria secondarily. If the lungs are immediately involved the treatment is usually started with one or another of the newer antibiotics such as aureomycin, chloromycetin, terramycin or streptomycin. This seems logical though penicillin is also indicated for the secondary bacterial invasion. Not many neck infections are preceded by this type of virus head cold onset, and in this minority the initial phase has passed before the neck is involved. During the height of the neck infection these newer antibiotics, other than penicillin, are rarely needed. By the time one ascertains the specific type of organism or organisms present, and sensitivity tests are made, the patient is usually well on the way to recovery. Occasionally I have used an aqueous solution of penicillin locally in the wound, introduced through an indwelling catheter which protrudes through the dressings and bandage. The nurse is instructed to inject five, ten or 20 cubic centimeters of the solution, containing 500 to 5000 units to the cubic centimeter, into the cath- or less arbitrary as it is non-irritating. I have not yet observed any allergic sensitization from this procedure.

In extremely severe and, perhaps, late cases when time-saving is important it may be advisable to add streptomycin to the penicillin therapy for a few days because streptomycin is more effective against gram-negative organisms.[8] Preparations combining penicillin and streptomycin are now available. None of the antibiotic drugs are wholly innocuous, and it may become necessary to discontinue administration and very carefully select substitutes. One must be constantly on the watch for evidences of toxicity which evidences must not be confused with the clinical manifestations of the infection or its complications.

### HORMESIS

This term is used to describe the stimulating effect of an antibacterial agent on the growth of otherwise sensitive organisms. It may also be used when organisms which are ordinarily not numerous or pathogenic become so when a pathogenic penicillin-sensitive or-

ganism is eliminated by penicillin therapy. The development of moniliasis is an example. Ordinarily the monilia organisms are not numerous or pathogenic, but may become so when the commonly present pyogenic bacteria disappear as a reult of the antibiotic therapy. The constituents of the bacterial flora normally present are thrown out of balance, and a severe unopposed monilia infection may result. This may occur anywhere in the alimentary canal and cause severe illness. Also, the natural antagonism of one type of pathogenic organisms, e.g. Gram positive, for another type of pathogenic organisms, e.g., Gram negative may be eliminated by the antibiotic. It is when the removal of the pathology in the portal of entrance, e.g., diseased tonsils, must be delayed for some reason, that there may occur relapses of the original infection after the infection in the neck has been drained and healed. Resumption of the antibiotic therapy in such cases may bring about these hormetic effects. One case in this series is an example of this tendency.

REPORT OF A CASE

Case 55, a 22 months old female child, was admitted to the hospital with a history of three days of upper respiratory infection consisting of acute nasopharyngitis, tonsillitis, an infected left lower molar tooth, gingivitis, acute bilateral tubo-tympanic catarrh, and cervical lymphadenitis on the left side. The patient was given penicillin and aureomycin by the family physician with prompt subsidence of all the inflammation except in the cervical nodes of the left side. These broke down and became fluctuant, and a large abscess was drained on the seventh day. The recovery was then uninterrupted, and the patient was discharged on the fourteenth postoperative day, the discharge from the hospital being delayed because of respiratory infection at home. This case is an example of what I have repeatedly observed, namely, that a lymphadenitis may go on to abscess formation after the infection has subsided at the portal of entrance under intensive antibiotic therapy. It is also an example of "no growth" culture reports from the abscess. The throat culture on admission was M. catarrhalis. Two weeks later the patient was readmitted to the hospital with recurrent infection of the upper respiratory tract, given antibiotic therapy and discharged eight days later. Several days later she developed a severe attack of chicken pox from which she made an uncomplicated recovery. When seen in the office a week later the tonsils were spotted with white patches of monilia which was attributed to hormesis. The patient was then readmitted to the hospital for intensive treatment so that the tonsils and adenoid could be removed. Penicillin was again given and the throat was swabbed three times a day with 1 per cent solution of gention violet. A marked improvement was immediately recognizable, and when the throat was comparatively clean the tonsils and adenoid were removed, following which the condition went on to complete recovery. Before and after the operation folic acid injections were given for neutropenia.

This case emphasizes the fact that the antibiotics may enable clinical problems to develop with which we were not familiar in the past.

In summing up the present and future status of the antibiotics I feel that I cannot do better than to quote directly from the last chapter of the Welch and Lewis book on Antibiotics.[8]

"Properly used these drugs are life saving, perhaps more so than any other agents available to the clinician. Improperly used they can be dangerous drugs. The inherent toxicities of certain of the antibiotics such as the eighth nerve damage caused by streptomycin, the nephrotoxicity of bacitracin, and the possible sensitization that may occur with any of them, are all obvious and can be controlled through judicious use. Other effects of these drugs are less obvious, and perhaps present more serious problems for the future. The development of tubercle bacilli resistant to streptomycin already portends a future public health problem. Indiscriminate use of this drug will accentuate this dangerous possibility. The development of increasing numbers of staphylococci resistant to penicillin and other antibiotic drugs is a problem that will increase in importance as time goes on. Perhaps more important than any of these is the ability of the antibiotics to stimulate rather than retard the growth of microorganisms in vivo."

## CONCLUSIONS

1. Blood stream infections have been reduced by the antibiotics.

2. Tracheotomy will still be necessary occasionally because of the frequency of the Ludwig's angina type of infection.

3. Pure cultures of streptococcus hemolyticus are now less frequent. The staphylococci and the alpha streptococci are more frequently present.

4. Moderate masking of symptoms and signs is to be expected under antibiotic therapy.

5. The visceral fascia type of infection has been reduced from 10 per cent formerly to 5 per cent now. Some of this reduction may be attributed to fewer infections from foreign bodies.

6. Lymphadenitis, with or without abscess, occurred in 56 per cent formerly and 43 per cent now—a moderate decrease.

7. The regions of involvement have changed from a predominance of pharyngomaxillary, or parapharyngeal type formerly to a predominance of submaxillary, or Ludwig's angina, type now.

8. The portals of entrance have changed from a predominance of pharynx and tonsils formerly to a predominance of dental and salivary glands now.

9. It is not yet time to throw away our scalpels, and I feel it never will be.

421 HUGUENOT ST.

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XLVIII

# CARTILAGE HOMOGRAFTS IN RHINOPLASTY: A CRITICAL EVALUATION

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CHICAGO, ILL.

Of all the materials that have been employed for the correction of defects in the contour of the nose, none has stood the test of time as well as human cartilage. This does not necessarily imply that cartilage is ideal in every case nor does it mean that it is 100 per cent effective but, by and large, in the majority of cases it can be relied upon to accomplish a lasting result. Theoretically, the ideal material should be one that approximates as closely as possible the tissue it is supposed to supplant, should be capable of being retained without change in its new environment, should be readily obtainable and easily molded to the desired shape. Furthermore, it should possess sufficient density to withstand the normal minor traumas to which the nose is exposed, and should not be so rigid as to be unyielding under normal stress.

On the basis of the standards thus set forth, it is readily apparent that neither cartilage nor bone alone, and certainly no foreign body material, possesses all of these qualifications. The nearest approach to this ideal would be a composite autogenous bone and cartilage graft taken from the rib. While the difficulties of obtaining such a graft are not too great, the problem of shaping is more complex because of the difference in density between the two components. A smoothly sculptured contour is essential inasmuch as the least irregularity is visible through the overlying skin.

Autogenous costal cartilage alone is almost ideal but has certain disadvantages which must be reckoned with. The most annoying of these is its tendency to curl after being in situ for some time. Despite the most careful cutting and shaping, this phenomenon makes its appearance in a certain percentage of cases within a few weeks to a number of months. In an effort to prevent this complication New and Erich[1] suggested boiling the cartilage. This pro-

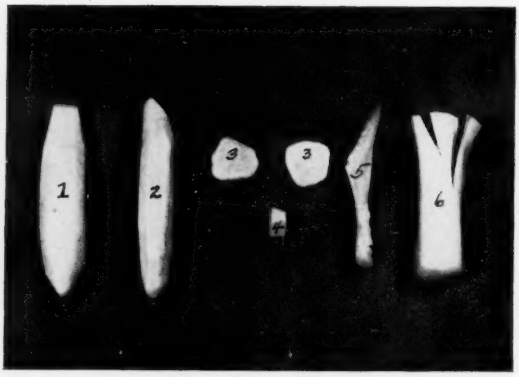


Fig. 1.—(1) Dorsal implant, top view; (2) side view. (3) Thin discs cut from full thickness of rib. (4) Small "button" for implantation into nasal top. (5) Strip showing tendency to bend. (6) Full thickness of rib cartilage sliced to show tendency to curve in direction of cortex.

Fig. 2.—Section of full thickness of rib showing differing densities of cortex and medulla.

cedure, however, means that the viable cells are destroyed, thus depriving the implant of its viability and losing any chance it might have had to survive as a living structure. It reduces the implant to the status of refrigerated cartilage and makes the resection of a rib an unnecessary operation.

My observations on the tendency of costal cartilage to curl lead me to the conclusion that this phenomenon is due to the difference in density between the cortical and medullary portions as well as to the persistence of fine strands of perichondrium. This can be easily demonstrated by slicing off a thin strip from the cortex of a piece of rib cartilage and noting how promptly it forms an arc in the direction of the denser layer (Fig. 1 and 2). How to minimize this tendency is a matter that I shall discuss later in the paper.

Another difficulty with cartilage lies in the texture of the particular piece in question. Cartilage of uniform density is extremely difficult to obtain, since it is found only in young well-developed adults. Cartilage obtained from children and adolescents is incompletely chondrified and, being partly fibrous, is more likely to be absorbed. On the other hand, cartilage obtained from adults approaching middle life and beyond is partly ossified and experience has shown that the lacunae of the ossified portions are soon invaded by tissue juices and various wandering cells which absorb a portion of the implant, leaving conspicuous irregularities in contour.

The fact that our preference is for young costal cartilage does not imply that septal cartilage is not equally desirable. Peer[2] has shown that this material will retain its shape for long periods of time. However, the chief shortcoming of septal cartilage is the fact that its bulk is not adequate for most of the saddle noses we have seen. There is no question that for minor defects it is a most suitable material.

In addition to the items just mentioned one has to take into consideration also the possibility of postoperative infection and foreign body reaction. These complications, though comparatively rare since the widespread prophylactic use of antibiotics, are nevertheless a possibility which must be recognized. In the few cases which we have encountered it has been our impression that infection was not due to lack of sterility of the implant or the solution in which it was kept, but rather to the presence of pathogenic organisms in the operative field. One must, therefore, be extremely careful to avoid

operating on a patient who has recently suffered an acute upper rspiratory infection, or in the presence of an active sinusitis.

In a further effort to avoid contamination from within the nasal cavity, it has been our practice in most cases to introduce the implant through an incision in the midline of the columella near the tip of the nose. The advantages are that the field can be completely sterile, which is not always the case with intranasal incisions, and the pocket which receives the implant is completely shut off from the intranasal cavity. Another advantage of this approach is that the pocket can be tunnelled more accurately since it is approached directly in the midline, whereas an intranasal incision must be in one or the other vestibule, which involves displacing the tip in order to maintain the midline dissection. If the incision is accurately placed in the midline and carefully sutured without tension, the resulting scar will be scarcely noticeable even on close inspection.

As for foreign body reactions, it is difficult to account for this occurrence inasmuch as in the instances where they were observed, the cartilage came from the same batch where the preponderance of successful takes would indicate that intolerance was not due to the cartilage itself. It is significant to note, however, that foreign body reactions were more common in columellar implants than in the dorsal. I believe this can be accounted for by the fact that the columella is subjected to greater stress than the nasal dorsum, due to traction of the upper lip in talking and eating. Also, the columella is more exposed to trauma. These factors also account for the more frequent absorption of columellar struts than is the case with dorsal implants. Stress and tension due to the scars of previous surgery or trauma will certainly predispose to rapid absorption of a cartilage implant in most cases.

The situation as regards dorsal implants is different. Here a properly shaped implant that is not introduced under tension can become firmly attached to the underlying bone and remain intact for an indefinite period of time. I have found it advantageous to elevate the periosteum over the nasal bones up to and beyond the nasion and to roughen the bone with a rasp. A firm fibrous union takes place between the roughened bone and the cartilage, which fixes it so that it is immobile. At the same time the lower portion of the graft which is less firmly attached is capable of being bent from side to side in conformity with movements of the mobile portion of

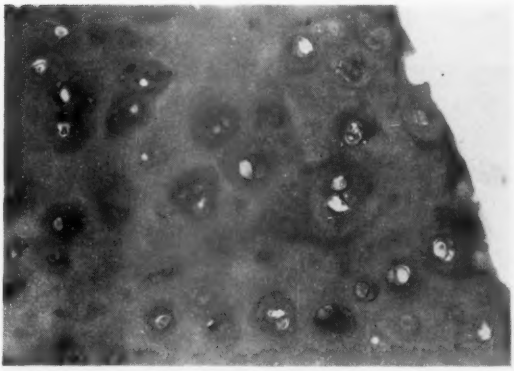

Fig. 3.—Cartilage removed after having been in situ over five years.

the nose. This makes it more desirable than a bone implant, which is too rigid if it is long enough to extend below the lower end of the nasal bones. Furthermore, experience is beginning to accumulate to show that a bone implant which is not in contact with bone is likely to be absorbed.[3]

With regard to the prevention of curling, I have found this complication extremely rare when the implant is cut from the core of the full thickness through its long axis, thus balancing the cortical and medullary portions. This is illustrated in Figure 1, which also demonstrates how the upper end of the graft is bevelled so as to fit closely into the curve of the nasion.

In order to insure fixation of the implant exactly in the midline, it has been my practice to apply mild pressure with a well-padded molded copper splint for a period of at least four days.

As to permanence of these dorsal implants, I have observed cases for as long as twenty-five years without noting any appreciable diminution in their size or shape. Examinations of specimens removed after a long period of time show a remarkable degree of preservation of the cartilage stroma despite loss of viability of the cells (Fig. 3).

In this connection, the case reported by Brunner[4] is particularly significant, at least to me. I recognized the photos as those of a

Fig. 4.— Patient operated upon in 1924.

Fig. 5.—Same patient re-operated upon in 1946 (Brunner).

patient upon whom I had operated in 1924, and I am grateful to him for permission to reproduce them (Figs. 4 and 5). Brunner reported the histologic status of the cartilage which I had implanted and which, for good reasons, he found it expedient to remove. He found the columellar portion in a good state of preservation, whereas the dorsal portion was broken up and partly absorbed. It may be that his explanation of the latter circumstance is correct, because it is likely that with the passing of years the nose may have been the site of some trauma. However, it has been my experience that dorsal implants when properly affixed to the underlying abraded bone survive much longer than do the columellar.

In addition to its use in the nasal dorsum and columella, I have found that small bits of cartilage can be successfully employed to fill minor depressions which seem to annoy many patients (Fig. 5). In these cases it is best to use thin slices taken from a cross-section of the whole cartilage. Even though such thin segments are likely to be absorbed, experience has shown that the fibrous tissue which replaces them is adequate for the maintenance of the desired contour. I have used implants of this nature to restore the contour of the alae and columella where irregularities have resulted from trauma or previous surgery. I have also found a small segment of cartilage useful in filling out a flat nasal tip as described by Sheehan.[5]

In conclusion, I wish again to emphasize the points which were elaborated in the paper and which will make for a higher percentage of successful takes. These are:

1. Employment of young adult cartilage only.

2. Proper cutting and shaping of the implant.

3. Scrupulous care in the preparation of the bed which is to receive it.

4. Under no circumstances to introduce the implant under tension or in areas where excessive scarring is likely to cause pressure, thus favoring absorption or distortion of the graft.

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Fig. 6.—Irregularities in alar margin from previous surgery filled in with thin discs of cartilage.

Fig. 7.—Distortion due to infection: corrected by single dorsal implant of cartilage.

Fig. 8.—Distortion of columella and nares due to trauma: correction by repositioning of lower end of septum. Cartilage implant entire length of columella introduced through the horizontal scar which was opened and resutured.

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XLIX

# VACUUM FRONTAL SINUSITIS—ABSORPTION OF AIR FROM THE FRONTAL SINUS

## AN EXPERIMENTAL STUDY ON DOGS

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The medical literature contains scant information as to what happens to gases trapped in the middle ear by closure of the eustachian tube or in a paranasal sinus by closure of the sinus duct. Sluder and many others have written about a "vacuum headache" which presumably is due to a relative vacuum in the diseased paranasal sinus but the literature contains no sound observations on the possibility of such a condition. On theoretical grounds it would seem that air could be absorbed via the blood stream as in the case of atelectasis and other conditions. Hilding has proposed a theory that ciliary action in the sinus ducts or eustachian tubes could remove quantities of mucus and encased air.

The frontal sinuses of dogs were chosen for the experiments because of their relative large size; an important factor when ease and accuracy of gas measurements in a closed cavity are essential. What was needed was the following: 1. An air-tight closure of the duct leading from the sinus to the nose and 2. Another air-tight connection between the closed sinus and a manometer which could measure changes in volume and pressure of the gases trapped within the sinus.

The precise procedure followed is given below but a prior word concerning the most difficult problem, technically, encountered might

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be given. The problem of securing an air tight obstruction of the nasofrontal duct was difficult to solve. Obstruction of the duct from the sinus end required a sufficiently solid plug to preclude any movement of mucous or other secretions by ciliary action as well as being air tight. None of the various substances tried such as petrolatum-saturated gauze packs, dry gauze packs, liquid paraffin dropped into the duct orifice, or a shaped soft rubber plug inserted into the sinus end of the duct to form a tight seal could be done successfully. Either bleeding occurred when the plug was forced into the duct or the plug was not sufficiently tight to prevent the passage of air. Blood in the sinus was undesirable because possible diffusion of the gas in the sinus directly into the capillaries would be altered and the blood itself would cause volume changes.

It was finally found most feasible to drill a small trephine hole through the nasal bones over the naso-frontal duct just inferior to the floor of the sinus and to obstruct the duct by external pressure by packing through that portal. It would have been ideal if the duct could have been obstructed by dissecting it out and then ligating but this proved technically impossible. A satisfactory arrangement, detailed below, was found by snuggly inserting dry gauze packing through the trephine hole directly on top of and around the naso-frontal duct until it was completely obstructed.

The experiments were made on 15 dogs in which 23 different frontal sinuses were used. The average weight was 22.3 pounds and the average volumes of the frontal sinuses were for the right and left respectively, 3.1 and 2.6 cc. Each animal, before being used, was carefully inspected for evidence of disease. Particular attention was paid to the respiratory tract when searching for evidence of disease. Anesthesia was secured by the intravenous injection of 6.5% nembutal until a sufficient depth of anesthesia had been reached. From time to time additional amounts of intravenous nembutal were needed to maintain the anesthesia. The animals were observed from 60 to 240 minutes before terminating the experiments.

In preparing the animal after anesthesia had been obtained a trephine hole was made on the animal's nose over the nasofrontal duct. The hole was approximately 1 cm. in diameter and usually was located approximately ½ cm below the floor of the frontal sinus. In removing the bone the mucous membrane was not excised as occurs when a trephine opening is made into the frontal sinus (de-

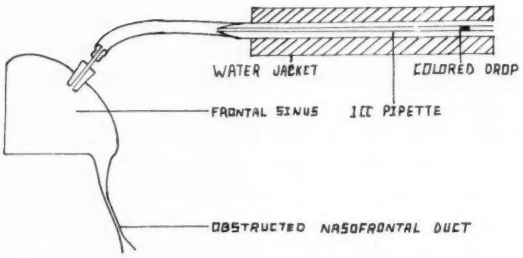


Fig. 1.—The apparatus used to measure volume changes.

scribed below). Rather the mucous membrane was elevated from the under surface of the nasal bone and the ascending process of the maxilla over an area corresponding to that occupied by the fronto-nasal duct. Obstruction of the duct was done by packing ½ inch gauze snuggly around and on top of the naso-frontal duct.

A second trephine opening of approximately 1 cm in diameter was then made through the anterior bony and mucosal wall of the frontal sinus, picking the highest dome of the sinus as the site for the hole. As soon as entry to the sinus was gained a careful inspection for blood was made and if more than a little blood-tinged fluid was seen the experiment was terminated. If the sinus was clean of blood a rubber stopper carrying a No. 16 blunt needle in its center was immediately fitted snugly into the hole. The needle was then connected by means of rubber and glass tubes to a 1 cc pipette graduated in 1/100s. This pipette was held horizontally on a level with the sinus and contained a colored drop of alcohol as an indicator. This volumetric scale was insulated against changes in volume produced by variations in room temperature by enclosing the horizontal 1 cc pipette (except for the tip which received the rubber connection) in a water jacket.

A closed system of the frontal sinus, connecting tubes, and horizontal 1 cc pipette was thus created and any change in volume of the gases within the sinus was reflected in the motion of the colored drop.

After conclusion of the measurements the efficiency of the naso-frontal duct-block was tested by substituting a water manometer for

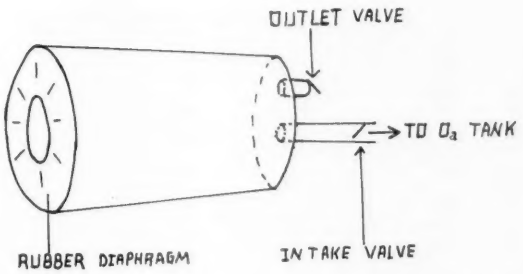


Fig. 2.—Diagram of the snout mask used during experiments when oxygen was administered.

the 1 cc pipette. Only those cases in which the block withstood the weight of 20-50 cm of water were included. The frontal sinus of the animal was finally opened and examined for blood, mucus or other contents. If time and the animal's condition permitted first one and then the other frontal sinus were tested.

There were three series of experiments, the first using the set-up described above, was designed to measure changes in volume. The second series of experiments was designed to measure changes in pressure. The apparatus necessary in this case was the same as that described above with the addition of a second manometer-tube, held vertically, interposed between the dog and the horizontally held 1 cc pipette. Then either of two things was done. Either (a) the volume was followed for a short period and when it began to fall indicating beginning reduction in the volume of gas the pressure tube was opened and the volume tube blocked off or (b) the pressure was followed throughout. The tip of the vertical pressure tube was immersed in water and measurements of the height in mm of the water column were made.

The third series of experiments was done using either the volume-measurement or the pressure-measurement apparatus. After following the animal for 15 to 45 minutes commercially pure oxygen was administered to the animal in an effort to reduce the $pN_2$ of the blood and thereby theoretically enhance the speed of absorption of the air trapped in the frontal sinus. The gas was administered by a snout mask containing an intake valve for the oxygen and an out-

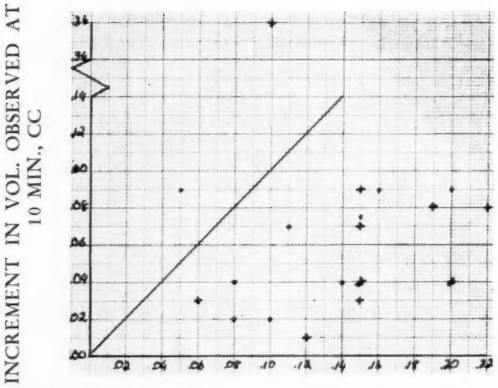


Fig. 3.—The + points indicate an experiment in which the volume continued to rise after the first 10 minutes, the black dots an experiment in which the volume fell after the first 10 minutes.

let valve for the expired air. On inspiration the former valve would open, permitting oxygen to enter the mask, and on expiration the latter valve would open, permitting expired air to be forced out. Thus the re-breathing of carbon dioxide was held at a minimum.

Post-mortem examinations were done on all the sinuses.

## RESULTS

1. *Volume changes in closed sinuses.*

Of the 23 sinuses studied the *volume increased in every case for the first* 10-20 *minutes.* To what extent this increase in volume of the trapped gases can be attributed to changes in temperature was investigated by calculations based on Charles' law which states that the volume of a gas varies directly with the absolute temperature, the pressure remaining constant. The air entered the sinus at room temperature but after closure of the trephine openings, thus trapping the air in the frontal sinus, the temperature of the trapped air rose to near that of the body temperature and, according to the law

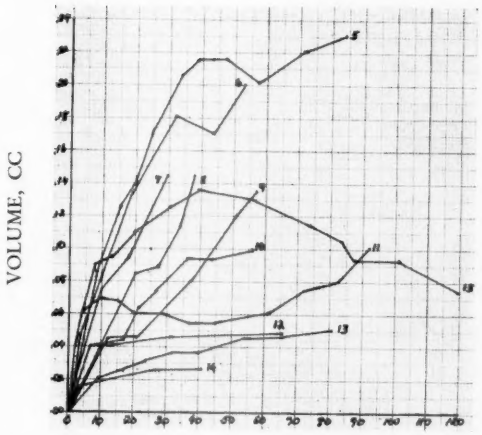


Fig. 4.—Experiments in the gas volume continued to rise after the first 10 minutes.

of Charles the volume must have increased. To calculate how much the volume changed the following formula was used:

$$V_1 = \frac{VT_1}{T} \times \frac{B\text{-}pH_2O}{B\text{-}p_1H_2O}$$

where $V_1$ the new volume of the trapped gases

V the original volume of the trapped gases

$T_1$ the absolute temperature of the trapped gases after they have increased to near body temperature

T the absolute room temperature

B the barometric pressure, assumed to be 740 mm mercury.

$p_1H_2O$ the partial pressure of water vapor at intrasinus temperature

$pH_2O$ the partial pressure of the water vapor at room temperature

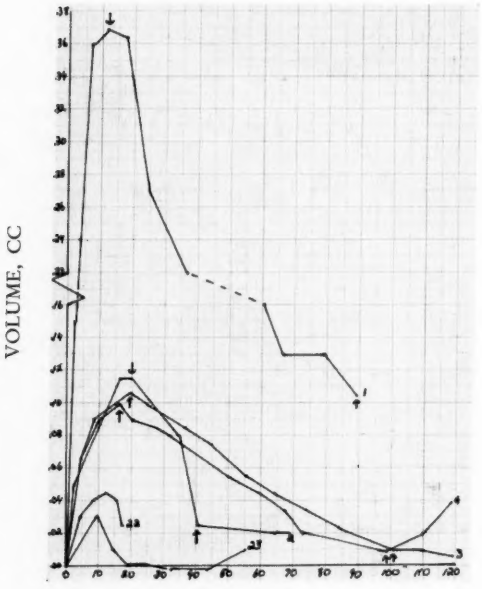


Fig. 5.—Six experiments in which a fall in volume was observed. The points between the arrows were used in calculating the mean rate of fall. (Also Exp. 21)

The intrasinus temperature was measured simultaneously with the rectal in four dogs and, after a period of approximately 20 minutes, was found to average 4°C lower. In the above calculations $T_1$ is taken as the rectal temperature minus 4°C. The value for the initial intrasinus temperature, T, is taken as room temperature. This is not strictly accurate, inasmuch as the initial intrasinus temperature probably lies somewhat above room temperature.

The observed increment in volume of the trapped gases at 10 minutes is plotted against the calculated increment due to the increase in the temperature of the gases. If there were a perfect equality between the observed increase in volume of the gases at the end of 10 minutes and the theoretical increase due to increased temperature of the gases the plots would fall on the solid line. The fact

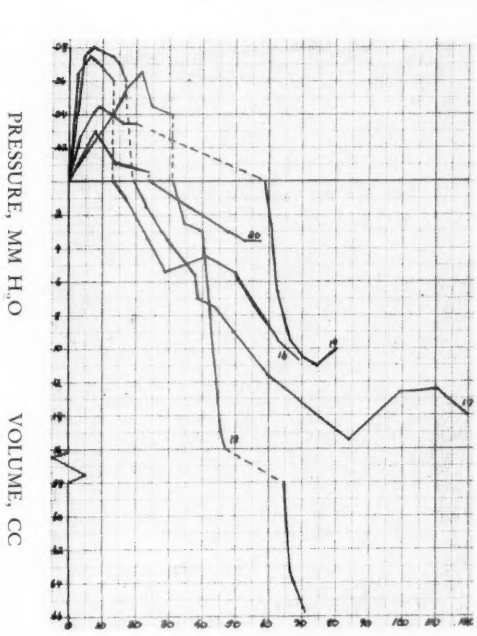


Fig. 6.—The five experiments in which a fall in pressure was observed.

that all but two of the 19 points are below the solid line indicates that although the temperature change must have contributed to this initial increase in volume other factors operating in the reverse direction may also have been involved. The error made in the calculations in assuming the initial intrasinus temperature to be identical to room temperature, as pointed out above, may be enough in itself to cause the points to fall below the solid line. If the initial intrasinus temperature were taken as midway between the room temperature and the final intrasinus temperature then the points do lie close to the solid line.

Following the initial rise in volume of the trapped gases, in 11 of the 23 sinuses experimented upon, the volume continued to rise for the duration of the experiment and in the remaining 12, the volume decreased. The courses of the volume changes are indicated

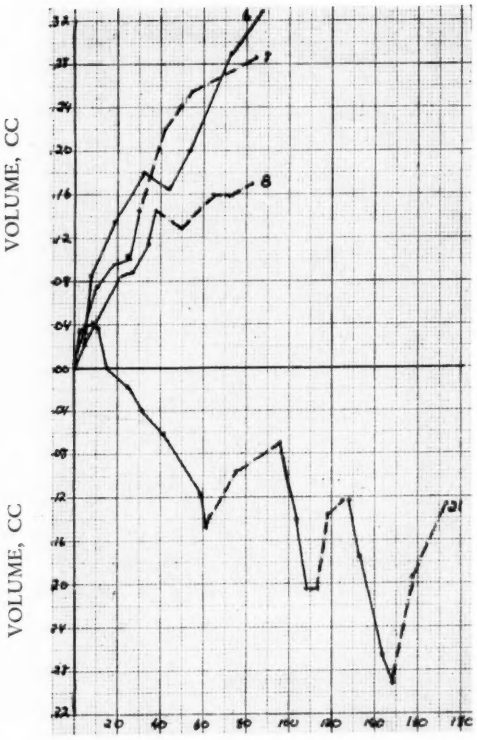


Fig. 7.—The effect of $O_2$ (dash) inhalation on the volume of the gases in the frontal sinus.

separately for those which continued to rise and those which reversed and began to fall.

In calculating the average rate of rise in volume the plots from 10 minutes to the termination of the experiment as given in experiment numbers 5, 6, 7, 8, 9, 10, 11, 12, 13, and 14 were used. The average rise in volume was found to be .001841 cc/min.

To obtain the average rate of decrease in volume plots between the two arrows in experiments numbers 1, 2, 3, and 4, and 21 were

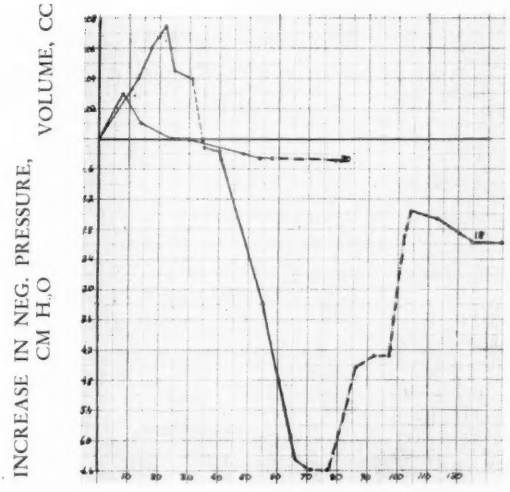


Fig. 8.—The effect of $O_2$ inhalation (dash) on pressure within the frontal sinus.

used, the average rate of decrease in volume was found to be .00252 cc/min.

Only one experiment, number 21, showed a reduction below the original volume. However, if the expansion of the trapped gases due to temperature changes is excluded 12 of the 21 showed a reduction below the original volume. In making this calculation the increase in volume due to the temperature change was subtracted from the observed volume at the time the volume measuremnts were terminated. Those experiments showing a fall below the original volume were numbers 3, 4, 11, 15, 16, 17, 18, 19, 20, 21, 22, and 23. Those not showing this fall below the original volume were numbers 1, 5, 6, 7, 8, and 10. Experiments 12, 13, and 14 showed essentially no volume change after the first ten minutes.

2. *Pressure changes in closed sinues.*

In the five sinuses experimented upon in this group the volume had begun to fall, before the volumetric apparatus was closed and the

TABLE E.

| EXPERIMENT NUMBER | NEGATIVE PRESSURE DEVELOPED $mmH_2O$ | RATE OF FALL IN PRESSURE mm/min |
|---|---|---|
| 16 | 10.5 | .184 |
| 17 | 15.5 | .250 |
| 18 | 66. | 1.65 |
| 19 | 11. | .687 |
| 20 | .35 | .010 |
| mean | 20.67 | .556 |

manometer opened. The detailed data are given in Table E. The greatest negative pressure recorded was in experiment number 18 and was 66. mm water. The average for the group other than experiment 18 was 9.34 mm water.

3. *The effects of oxygen inhalation on volume and pressure changes.*

In experiment number 6, 7, and 8 the volume of the gases trapped in the frontal sinus was increasing when the inhalation of oxygen was instituted. In none of these three experiments was the increase in volume of the trapped gases reversed by the inhalation of oxygen. This was contrary to expectations, since by inhaling oxygen the $pN_2$ is reduced and absorption of the trapped nitrogen should be thereby enhanced. In experiment 21 the volume of the gases had fallen below the original volume when oxygen-inhalation was started. Instead of the expected increase in the speed of absorption of the trapped gases, the volume change immediately reversed itself and began to increase. On the replacement of oxygen with air the volume again began to fall, only to increase again after the reinstatement of oxygen-inhalation.

In experiments 18 and 20 oxygen-inhalation was inaugurated after a fall in pressure was being recorded on the manometer. Rather than the expected further decrease in pressure, the pressure increased after the onset of oxygen-inhalation by the animal.

Post-mortem examinations carried out on each sinus experimented upon displayed a variable accumulation of small amounts of blood and mucus. In no case was a gross edema of the mucous membrane seen.

## DISCUSSION

From a theoretical standpoint air, or other gases, should be absorbed from a paranasal sinus where they have been trapped the same as in the cases of trapped gases in atelectasis, pneumothorax, pneumoperitoneum, subcutaneous emphysema and gaseous distension of the intestine. The evidence obtained in these experiments which indicates that there is a partial absorption by the blood of gases trapped in a dog's frontal sinus is as follows:

a. The values recorded for the observed increment in volume of the trapped gases at the end of 10 minutes, with two exceptions, are regularly less than the theoretically predicted values on the bases of increase in volume due to the temperature change. This indicates that some other factor, possibly absorption, was operating to decrease the volume of the trapped gases.

b. In 15 of the 21 sinuses reported in these experiments (No. 2 and No. 9 excluded because of incomplete data) the volume at the termination of the volumetric recordings was below the original observed volume. In five of these experiments the pressure was measured and found to be negative.

c. These observations suggest that gases were absorbed from the closed sinus cavities. The rate of absorption in five successful experiments (Numbers 1, 2, 3, 4, and 21) averaged 0.00252 cc/min or about 0.09% of the total volume per minute which is a rate considerably in excess of the ability of the lung to absorb gases as pointed out below.

There are several factors which indicate that absorption of gases trapped in a dog's frontal sinus should be a much slower process than occurs for example in the lung alveoli.

a. In the dog's frontal sinus the surface area available for absorption of the trapped gases is small compared to the lung alveoli. To calculate the surface area of the sinus it is assumed the shape of a sphere which would under-estimate the surface. Then, knowing

the average volume of the 23 sinuses examined to be 2.8 cc, the surface area of the average sinus is obtained as follows:

$$\begin{aligned}
\text{Volume of a sphere} &= 4.189r^3 \\
r^3 &= 2.8/4.189 = .666 \\
r &= .873 \\
\text{Surface of Sphere} &= 12.57r^2 \\
&= 12.57 \times .873^2 = 9.58 \text{ cm}^2 \\
\text{Surface of sinus} &= 9.58 \text{ cm}^2
\end{aligned}$$

Comparing this with the surface of the lung alveoli, 70 square meters (73), reveals how much more efficient the lung should be in absorbing gases.

b. The sinus mucosa is approximately 1 mm thick in contrast to the alveolar walls which are only one cell layer in thickness.

c. The sinus mucosa is relatively avascular compared to the alveolar walls. The result of b. and c. is that the average distance the trapped gases would have to diffuse is relatively greater than is the case of the gases contained in the lung alveoli. A comparison can be made here between the surface area of the lung alveoli related to the thickness of the alveolar walls (assumed to be .01 mm) and the corresponding figure for the sinus.
Alveoli:

$$\text{Area (A)/Thickness (t)} = \frac{A}{t} = \frac{7 \times 10^5}{.01} = 7 \times 10^7$$

Sinus:

$$\text{Area/Thickness} = 9.58/1. = 9.58$$

Thus the lung alveoli should be, on this basis, $7 \times 10^7/9.58$ $.729 \times 10^7$ times as effective in the absorption of gas as the sinus.

In experiments Numbers 1, 2, 3, 4, and 21 the observed average value for the absorption of gas was 0.00252 cc/min. This figure multiplied by the value representing the greater efficiency of the lung as compared to the sinus gives a product of 18370.8 cc/min which is far greater than the known capacity of the lung for absorption.

Thus though several factors have been pointed out which indicated that the sinus absorb gases slowly compared to the lung the observed values indicate the opposite.

There are a number of uncontrollable factors which may account for the inconsistency of the results.

a. The changing permeability of the sinus mucosa to the absorption of gases may be due to a variable covering surface layer of fluids, blood, mucus, etc.

b. Variable vasodilation and vasoconstriction may cause changes in the thickness of the sinus mucosa and thus decrease and increase respectively the volume of the sinus.

c. The presence or absence of secretions, hemorrhage, transudates in the sinus lumen, may alter the sinus volume.

The most surprising part of the work was relative to the respiration of commercially pure oxygen by the animal in an effort to influence the speed of absorption of the trapped gases from the frontal sinus. Theoretically the respiration of oxygen should have rapidly lowered the $pN_2$ of the arterial blood and thereby greatly increased the absorption of the nitrogen fraction (approximately 79%) of the trapped air. Instead of the expected result respiration of commercially pure oxygen lead to an opposite one. In those cases where the volume of gas was decreasing the inhalation of oxygen by the dog immediately lead to an increase in the gas volume. Similar unexpected results were obtained when a manometer was used instead of the volumetric apparatus. In this case instead of the theoretically expected accelerated fall in pressure there was a decrease in the negative pressure after the institution of oxygen respiration.

Possible explanations which relate to these unexpected results after respiration with oxygen include the following:

a. The possible diffusion of oxygen into the sinus lumen. If the inhalation of commercially pure oxygen by the animal increased the $pO_2$ of the blood perfusing the sinus mucosa above that of the oxygen in the trapped air there would be a diffusion of gas from the blood to the trapped air, thereby increasing the trapped gas volume. Against this possibility is the fact that tissue oxygen tensions are little affected by oxygen inhalation in the absence of arterial anoxemia, because the extra dissolved oxygen amounts to only a fraction of the

oxygen consumed by the tissues, so that venous oxygen tensions remain essentially unchanged. It is unlikely, although possible in principle, that the utilization of oxygen by the sinus mucosa is so small that it is bathed essentially by arterial blood: if this were true, oxygen inhalation might raise the mucosa $pO_2$ and permit diffusion of oxygen into the sinus cavity.

b. Possible changes might occur in the absorbing qualities of the surface of the mucous membrane. For example a sudden exudation of blood or serum concommitant with the inhalation of the oxygen would decrease the speed of absorption of the trapped gases as well as encroach on the sinus lumen. To explain the reversible effect of oxygen inhalation, a sudden absorption of this blood or serum would have to be visualized and this does not seem reasonable.

c. Possible changes might occur in the caliber of the mucosal blood vessels. If oxygen inhalation were in some way able to produce a reversible vasodilation in the mucosal blood vessels, with a resulting change in the mucosal thickness, the observed results could be explained. Perhaps this is the most likely explanation of the phenomenon.

The question arises as to whether the absorption of trapped gas in a paranasal sinus can explain the so-called "vacuum headache." In the five experiments in which pressure was measured an average -20.67 mm water was obtained and this does not seem of sufficient degree to produce a sinus pain of that nature.

## SUMMARY AND CONCLUSIONS

1. In 15 normal dogs under nembutal anesthesia a trephine opening was made through the anterior wall of the frontal sinus and the normal ostium occluded. Changes in pressure or volume of the gases enclosed within the sinus were then recorded from the trephine opening.

2. Of the 23 frontal sinuses in which volumetric measurements were made, all showed an initial 10 minute increase in volume of the trapped gases, but this increase was of less magnitude than expected on the basis of the temperature change alone.

3. After this initial 10 minute period the gas volume decreased in 12 of the 23 and increased in 11. The average rate of volume decrease was .002520 cc/min; the average rate of volume increase was .001841 cc/min.

4. Substitution of a manometer for the volumetric apparatus in five cases where the volume was falling indicated the development of a negative pressure. The greatest negative pressure recorded was 66. mm water and the average for the group was 20.67.

5. These results indicated that if absorption of trapped gases occurred in the sinus, the rate was very slow and only very small negative pressures developed. However, even this slow rate was greater than expected on the basis of a comparison with the lung. The inconsistent behavior indicates that other processes, such as vasomotor changes, secretion, bleeding, edema probably complicated the results.

6. The inhalation of 100% oxygen instead of accentuating the absorption of gases as expected, produced a readily reversible effect interpretable as either an increase in volume of the trapped gas or as a decrease (vasomotor) in volume of the sinus cavity.

L

# SURGICAL TREATMENT OF CHRONIC FRONTAL SINUSITIS

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It is my purpose in this paper to bring before you a method first described by E. C. Sewall[1] in 1935 at the Western Section of the American Laryngological, Rhinological and Otological Society meeting and subsequently published in the Archives of Otolaryngology in June, 1935, for the cure of chronic frontal sinusitis. I believe all are agreed that the handling of the nasofrontal duct is the greatest obstacle in the cure of this disease.

Many procedures have been described, such as mucous membrane flaps, skin grafting the newly made opening, the use of various tubes to be retained in the new opening for various lengths of time, such as the Ingalls[2] gold tube, the use of rubber tubes left in for various lengths of time, the use of tantalum foil as described by Goodale[3] and the acrylic obturator as described by Erich and New.[4] Also mention should be made of the work of Walsh[5] in which he described a method whereby the nasofrontal duct is left intact. However, we believe that in chronic frontal sinus disease, at least, the anterior ethmoid sinuses are diseased and, if so, they should be removed. With their removal it certainly is a difficult problem to leave the frontal duct intact.

Brown[6] in 1941 mentions the mucosal flap of Sewall and states, "Mucosal grafts as advocated by Sewall are of distinct advantage."

Lothrop[7] thirty-seven years ago stated, "Frontal sinusitis is caused and kept up by infection and obstruction; but if very free drainage is established and the sinus is made easily accessible to intranasal treatment all fistulas will close, all subjective symptoms will disappear, and the discharge from the sinus will cease or will be reduced to a minimum."

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Kernan[8] states, "The most popular and successful operation is one that removes the floor only, without disturbing the anterior wall, which in a great measure eliminates the possibility of osteomyelitis of the skull. To Jansen[9] belongs the credit of first describing in 1894 the cardinal principles and technique of such an operation. Knapp,[10] in 1899, reported a successful case, but it was Lynch in 1921[11] who vigorously put forward the advantages of this type of operation. Sewall,[12] in 1926, published his technique which has been amplified and refined by Ferris Smith,[13] and others."

However, it was not until 1935 that Sewall published an article in which he described in detail a method of using a mucoperiosteal flap to insure a permanent opening into the operated frontal sinus. Very little mention in literature is made of this procedure except to state that it had been tried but had been found unsuccessful. I hope to show in reporting fifty-seven successful operations done by the staff of the University of Oregon Medical School, and twenty of these were operated by myself, that in our hands it has been very successful. In fact, all have been symptom free following the operation. My oldest case was operated upon in the summer of 1935. He was seen by me a few months ago and was symptom free and had a good opening into both frontal sinuses. Some of you have seen a motion picture of this procedure which I showed at the American Academy in Chicago in 1950.

R. L. Goodale has stated, "After reviewing one hundred and ninety frontal sinus operations that the following seemed to be the cause of failure in the Lynch type of operation:

"1. Complete removal of the floor of the frontal sinus,

2. Removal of the spine of the frontal bones,

3. Removal of enough of the ascending process of the superior maxilla to give access to the anterior ethmoid region.

4. Complete ethmoidectomy and opening of the sphenoid sinus for drainage if the latter is involved. But even where this technique had been observed, a large number of operations had been failures because of adhesions. The prevention of scar tissue at the site of the nasofrontal duct is difficult to obtain. This region is involved in the ethmoidectomy so that the nasofrontal duct is removed as an anatomical structure. The area becomes merely a wide open passage."

I would add that a thorough removal of the frontal sinus lining is also essential.

As has been said before, probably the greatest obstacle to the cure of chronic frontal sinus disease is the problem of handling the nasofrontal duct, and, in my opinion, all efforts to preserve the nasofrontal duct are the chief causes of failures in frontal sinus surgery.

We prefer local anesthesia. One hour before operation we give nembutal 1½ grains. One-half hour before operation, we give morphine sulphate ¼ grain and atropine sulphate 1/150 grain. Cocaine crystals and adrenalin 1-1000 are brushed throughout the nasal cavity. Then we use regional anesthesia consisting of one or two per cent novocaine into which is added ten drops of adrenalin to the ounce.

Sewall advocated the complete removal of the duct and an opening made large enough and covered by a mucoperiosteal membrane from the nose to prevent granulations from forming causing adhesions, which are the causes of failure.

True, it is an operation that must be done with the utmost care in order to preserve the flap; it is a time consuming operation because to save the mucoperiosteal membrane for a flap and to remove every vestige of the floor and the sinus lining, especially in a large or shallow sinus, taxes the ingenuity of the surgeon. It, however, can be done. Also it can be accomplished successfully on cases in which sinus surgery has been previously done.

Most writers speak of curetting the lining from the sinus but this should never be done. The lining should be separated from the bone by dull periosteal elevators. It is desirable to have remote portions of the sinus cavity obliterated by granulation tissue and this cannot take place unless the original lining is entirely removed. If one is not sure that every portion of the membrane has been removed, picric acid-acetone solution may be used. This consists of 5 per cent picric acid in 35 per cent acetone solution, as described by Ferris Smith[13] in 1934. This toughens the membrane and makes its removal much easier. It is our belief that at least the anterior ethmoid sinuses and usually all the ethmoid sinuses are diseased where we have chronic disease of the frontal sinuses. Consequently we advise removal of at least the anterior ethmoids in order to obtain satisfactory results.

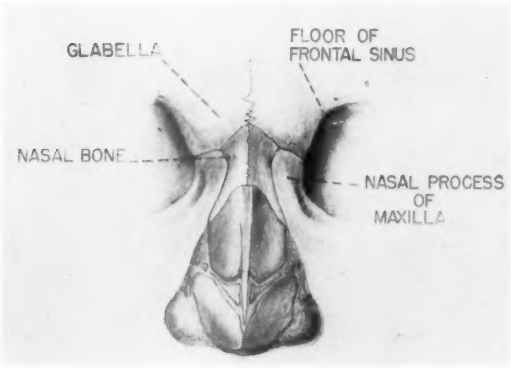


Fig. 1.—Anatomy of areas from which mucoperiosteal flap is obtained.

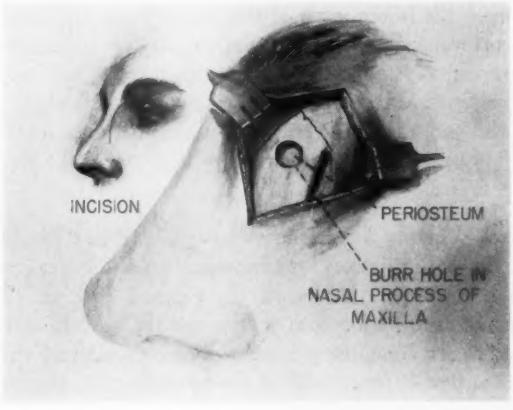


Fig. 2.—Showing incision and the burr hole uncovering the mucoperiosteal membrane.

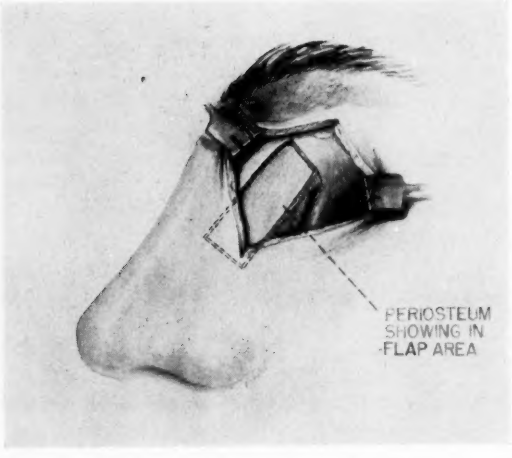


Fig. 3.—Showing the entire mucoperiosteal flap uncovered.

In performing the fronto-ethmo-spehnoid operation, the following technique should be followed. The skin incision is a curved one, following the inner angle of the orbit extending not farther than the supra-orbital notch and downward on the nasal process of the maxillary bone. Usually the incision is from 3 to 6 cm in length, which gives sufficient room because of the flexibility of the soft tissues. This usually suffices. If insufficient, an incision may be made at the outer margin, in sinuses which extend to the outer margin of the orbit.

However, in my experience I have never had to use the second incision. If, in the sinuses which extend to the outer margin of the orbit, the narrow part is thoroughly cleaned and the floor is taken entirely down, the periosteum will allow the soft tissues to push up and obliterate any remaining space. The elevation of the periosteum exposes the antero-inferior wall of the frontal sinus, the frontal process of the maxillary bone, and a portion of the nasal bone. The lacrimal sac is now raised from its bed, and the periosteum of the lamina papyracea is separated for a few millimeters. At this stage one should identify the anterior ethmoid artery and it should be tied and cut. This can be done by grasping the artery with long pointed forceps

Fig. 4.—Showing the cut flap and the beginning of frontal sinus operation.

and tying, as is done in any deep cavity, or by using some tying instrument such as the ones described by Sewall or Ferris Smith. The bony window is started just below the nasal fissure which is the suture joining the anterior wall of the frontal with the superior borders of the nasal bone, nasal process of the superior maxilla and the superior process of the lacrimal bone. The bone, which uncovers the mucoperiosteal flap and which is principally beneath the nasal process of the maxilla, but usually extends a millimeter or two onto the nasal bone, may be now removed with a mastoid gouge by carefully shaving down to the periosteum, making an opening large enough to use rongeurs to enlarge the opening. However, we found in our cadaver work that it is much simpler to remove the bone to make the bony window at least at the beginning, with a large sized cutting electrically driven burr. This is much faster and there is very much less danger in tearing the flap.

After the bone is removed for a few centimeters, the membrane or the periosteum, beneath the bone, is carefully separated a few millimeters at a time, and the overlying bone removed with rongeurs and the flap uncovered. The membrane is separated by passing variously curved periosteal elevators downward as well as laterally and upward to the nasal fissure to obtain a flap large enough to cover the inside of the glabella. At this point in the operation, two vertical incisions

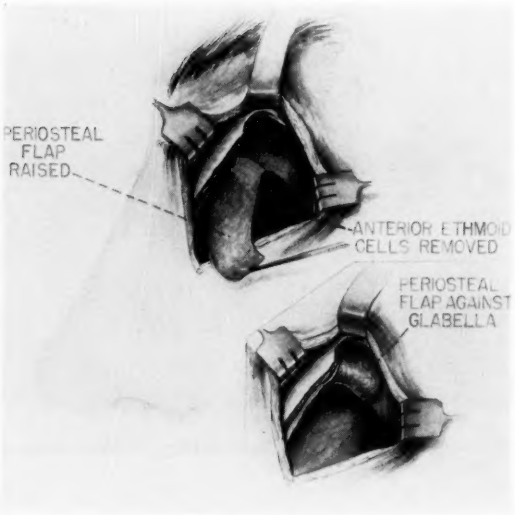


Fig. 5.—Showing the final step of the removal of the floor of the frontal sinus, the lining removed and the mucoperiosteal flap hinged at the nasal fissure and then turned into the glabella.

are made downward from the nasal fissure, wide enough apart to make a flap of the proper width. The horizontal or lower edge of the flap can now be cut by going through the nostril which is the inferior border of the flap. This sturdy piece of mucoperiosteum from 1 to 3 cm in width and from 3 to 6 cm in length is to be turned upward to line the medial wall of the new nasofrontal opening and more or less of the frontal sinus. This flap will now cover the inside of the glabella after preparation and prevent the formation of granulations and so prevent adhesions, which may cause closure of the new frontal nasal opening. At the same time it will extend into the sinus, and epithelium will grow out from it, covering the granulations that are formed within the sinus cavity. This flap is now pushed to the front of the nose and the fronto-ethmo-sphenoid operation started. The bony window is now enlarged upward, removing all of the heavy cancellous bone of the lower inner part of the glabella. If this is not removed, the cancellous bone will be an ideal place for the formation of granulations and consequently close the newly

made duct. The floor of the frontal sinus is thoroughly removed, leaving absolutely smooth edges and no projecting shoulders or spicules of bone. The success of the operation depends on: first, the removal of the entire floor; second, the removal of every particle of the mucosal lining; third, the removal of the diseased ethmoid cells; fourth, the removal of the cancellous bone of the glabella; and fifth, the making of a muco-periosteal flap of proper size. The diseased ethmoid sinuses, under complete visualization and accessible in any direction back to the spenoid wall, are now removed carefully. After completing this part of the operation, it is fairly easy to remove the anterior wall of the sphenoid and remove its entire lining.

The mucoperiosteal flap, which has been pushed aside, is now turned upward to line the medial wall of the new frontal opening and is kept in place either by a cigarette drain or a soft rubber tube extending into the nose to hold the flap in place. The skin incision and the periosteum is closed by a few interrupted dermal or silk sutures, which are removed about the fifth or sixth day. However, if you prefer, as some do, the periosteum edges may be closed first by catgut sutures and then the skin incision closed. The tube holding the flap in place should be removed about the fourth or fifth day and the skin sutures removed at the same time. It is well to remember that gauze of any type should not come in contact with the flap or it will be apt to adhere and remove the flap along with the tube. If the flap has been well made and of sufficient size, the after care is very simple and the new frontal opening will remain open permanently. We have found that in all sinus surgery the less we do in after treatment, the better. Let the tissues recover with very little washing. Crusts may have to be removed a few times and the nose may be cleaned by gentle suction rather than by irrigations.

The original dressings used by us consists of painting the closed incision with tincture of benzoin compound, then cotton placed over it and more tincture of benzoin added.

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LI

# RECURRENT LARYNGEAL NERVE PARALYSIS

## A REVISED CONCEPTION BASED ON THE DISSECTION OF ONE HUNDRED CADAVERS

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The purpose of this paper is to present a revised conception of the causes and mechanism of recurrent laryngeal nerve paralysis. The explanation is based on the fact that, at times, these nerves divide into two or more branches prior to entering the larynx. This division may occur at any point in the recurrent course from the clavicle to the cricothyroid articulation. The statement that the recurrent laryngeal nerve has been found to divide in the extra laryngeal areas could not be news to those familiar with the literature on this subject. This is a report on the dissection of 100 cadavers. It confirms the division of the recurrent laryngeal nerves in the extra laryngeal space. The information obtained from these 100 dissections not only offers a simple, practical and tenable explanation of the elementary and bizarre type of traumatic laryngeal paralysis but should assist in avoiding these paralyses.

There is experimental evidence[25, 26, 27, 28] to show that the initial division of this nerve produces a separation of the abductor and adductor fibers. It is logical to believe that since this separation occurs in an area that is commonly subjected to surgical intervention one should anticipate that there will be occasions when one or the other of these divisions will receive damaging trauma or actual severance. The relative vulnerability of the divisions will be influenced by their respective anatomical relationships. Clinical findings demonstrate that a certain percent of patients who have undergone thyroid surgery or other major neck surgery do have laryngeal muscle paralyses as a result of nerve injury. The importance of this fact

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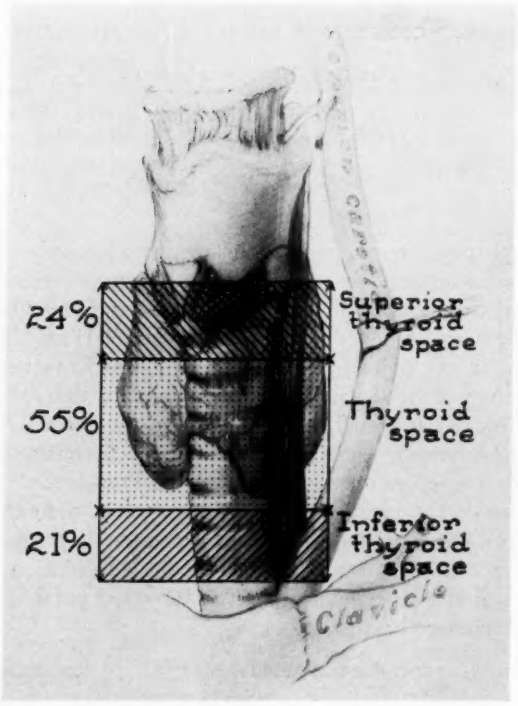


Fig. 1.—An arbitrary division of the extra laryngeal area into Superior Thyroid and Inferior Thyroid Spaces. The location of the divisions of the 86 nerves is shown in relative percent.

was not recognized nor the significance appreciated until recently.[44] The general acceptance of this knowledge will come slowly but surely. It is hoped that concurrently with its acceptance will be the recognition of one further point, namely, that there are at times secondary and tertiary divisions of either the abductor or adductor trunks in the same vulnerable area (Figs. 3 and 4). The presence of these subsequent divisions offers adequate explanation for those instances wherein only some of the laryngeal muscles show evidences of paralysis. Such previous explanations as were produced were postulates, based on theory and could not be confirmed by controlled experiment, dissection or microscopic section. The demonstration of secondary and tertiary nerve divisions in the extra laryngeal areas offers an explanation that satisfies all the criteria for proof.

It was illuminating to find that the main trunk of the recurrent laryngeal nerve divided bilaterally prior to arriving within ½ cm of the cricothyroid articulation in 24% of the cadavers, and that either the right or the left nerve was found similarly divided in 43% of the dissections. It is also of interest to note that of the divided nerve trunks, such divisions were not confined to the right side, contrary to the statement of Meckel[4] in 1832. This is evident in the instances of bilaterally divided nerves. In the cadavers showing unilaterally divided nerve trunks there is almost equal distribution of division on the two sides. The exact figures show a ratio of 20 on the right side as compared with 18 on the left side. A summation of these figures shows that of the 200 nerves, 86 or 43% were either unilateraly or bilaterally divided nerve trunks. If one deletes those that divide high in the superior thyroid spaces, some 21 in number, there still remain 65 examples of divisions in areas frequently invaded during neck surgery. In other words, more than one-third of the patients can be expected to have one or more divided nerve trunks in the thyroid or inferior thyroid spaces and therefore one-third of the patients carry potentials for injury of one or the other of the main trunk branches of the recurrent laryngeal nerve.

The fact that the recurrent portion of the laryngeal nerve divides in the extra laryngeal area in more than one-third of the dissections has been conclusively demonstrated on a purely anatomical basis. The application of this knowledge has a practical value.

Paralyses of the larynx have been a controversial subject for many years. The literature is bulky and richly endowed with con-

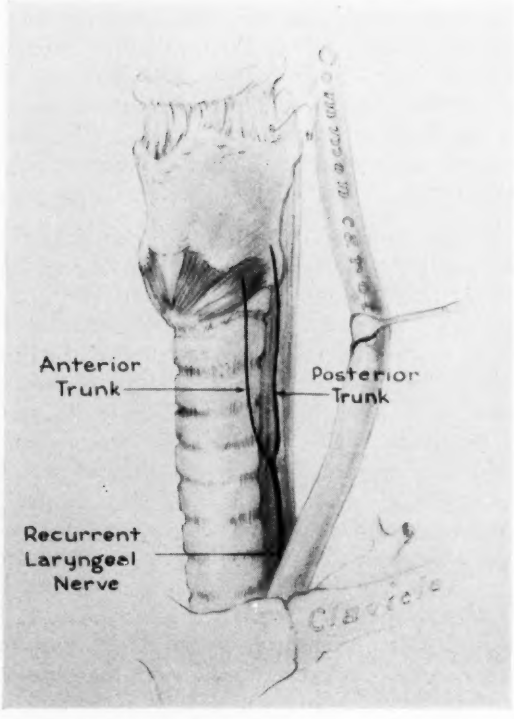


Fig. 2.—A simple divided left recurrent laryngeal nerve.

tributions that are of little value. There are many demonstrations of how active the mind can be in presenting pseudo-logical, abstract, or philosophical deductions, even though the explanations had no counterpart in any other nerve muscle relationship. These are excellent examples of attempts to construct anatomy to fit a clinical picture. This is seldom a satisfactory approach especially when it entails establishing conditions peculiar to a single area which violate fundamental embryologic and physiologic principles. Conversely, several present lucid descriptions of their dissections to explain the clinical pictures.

The division of paralyses of the larynx into central and peripheral types is rather well established and accepted on the basis that upper motor neuron lesions produce spastic paralyses whereas peripheral nerve lesions or lower neuron damage produces a flaccid paralysis with ultimate fibrosis and contracture, following varying or variable periods of atrophy and fibrosis. The importance of this sequence of events is aparent but not too well appreciated, as far as the muscles of the larynx are concerned. A description of the accepted central origin of the fibers that make up the vagus nerve would tend to confuse rather than add to the purpose of this paper. There is a general agreement among authors and texts which is satisfactory in all respects.

The vagus nerve has the most extensive course and distribution of all of the cranial nerves. It is composed of visceral sensory and motor (para sympathetic pre-ganglionic) fibers as well as somatic and sensory fibers. The laryngeal paralyses of central origin are adequately described and presented satisfactorily. That portion of the vagus nerve, distal to the ganglion nodosum (ganglion of the trunk) does interest us. Our attention will be confined to two of the three branches of distribution that originate in the neck—the superior and recurrent laryngeal nerve. The third branch or group —the superior cardiac branches (rami cardiaci superiores: cevical cardiac branches) do not contribute to or interfere with paralyses of the motor or sensory function of the larynx.

The site of origin of the superior laryngeal nerves has not been a subject for comment or discussion. This does not hold true for the recurrent laryngeal nerve. Hérard[5] describes an anomalous origin of the right recurrent laryngeal nerve and in 1846 suggested "that we ought to be prudent in denying existence of the recurrent lary-

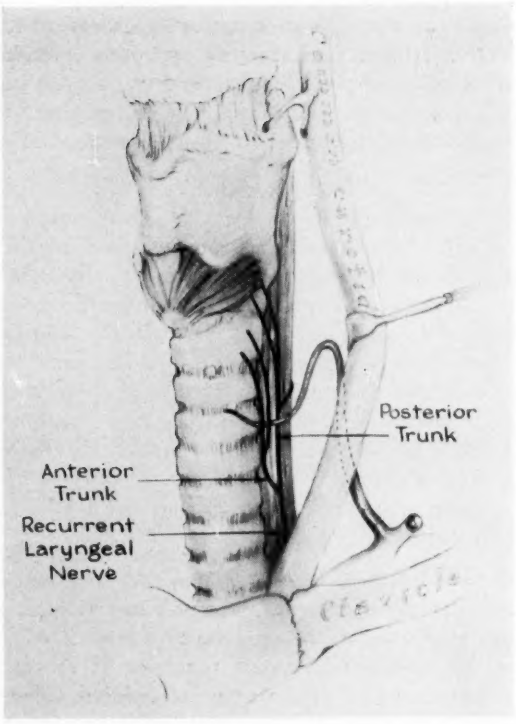


Fig. 3.—A four divisioned recurrent laryngeal nerve. In this specimen the anterior trunk of adductor fibers divided in the thyroid space anterior to the inferior thyroid artery. The posterior trunk of abductor fibers divided in the superior thyroid space posterior to the artery. A fifth branch is shown going to the esophagus.

geal nerve if we did not find it in its proper place." On rare occasions and in association with congenital developmental anomalies of the fourth pair of aortic arches wherein the right member atrophies, is misplaced or is absent, the right subclavian artery has its origin more caudad. Under these conditions the right recurrent nerve is cephalad to any structure which ordinarily would cause it to descend into the thorax with the subclavian artery and therefore does not have to assume a recurrent course to enter the larynx. This anomalous course of the right recurrent laryngeal nerve was first described by Steadman[2] in 1823. His explanation of the cause of this condition was not adequate. Hart[3] whose views are not wholly in accord with modern embryology, stated that the secret of the circuitous route was to be found in the natural law of embryologic or embryonic development. Reid[6] in 1847 gave the correct explanation of the origin of these anomalies. Several other authors[2, 3, 5, 6, 8, 9, 29] have reported additional examples of this anomaly. An excellent article by Work[29] covers the historical background, presents a typical case and refers the reader to Piersol[48] for the detailed explanation of the developmental factors which produce this condition.

Thus far, all cases wherein there has been aberrant origin of the recurrent laryngeal nerve, the anomaly has occurred on the right side. This may have induced several of the later investigators to make the statement that when the recurrent laryngeal nerve divides in the extra laryngeal areas, it only occurs on the right side. The question naturally presents itself as to what may occur in those rare individuals who have dextrocardia and transposed viscera. This question was answered in an article by Berlin[24] who cites Weatherford of Harvard, who, in 1934 presented findings to show that when such a condition occurs in these rare individuals it then occurs on the left side.

The historical background of interest in the function of the recurrent laryngeal nerve extends into antiquity. An outline of abstracts made while reading the earlier authors is incorporated under the heading "Chronological Data." It is offered for review by those who have covered this field, as a working and time saving reference for those who are less familiar with the literature and primarily in the hope that it will intrigue and perhaps stimulate others to find time to peruse this background when leisure permits or the conscience demands. Few authors who have presented reviews of the literature

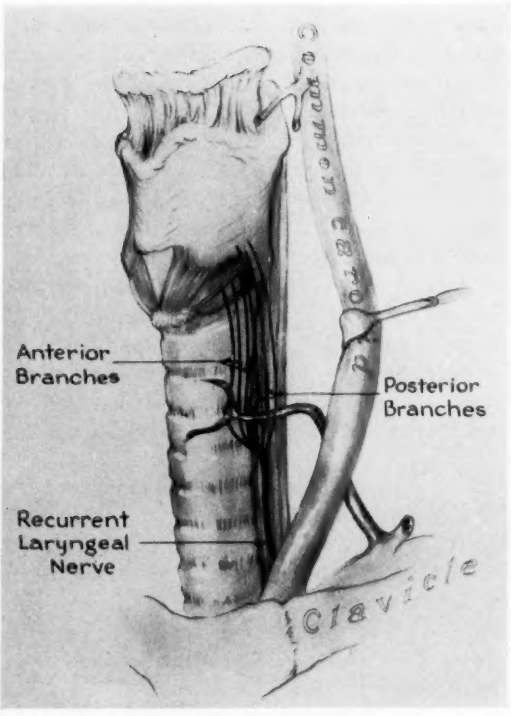


Fig. 4.—A six branched recurrent laryngeal nerve. This is an uncommon type of division in that usually the initial division into anterior (adductor) and posterior (abductor) trunks retain their integrity for longer distances before redividing. In this specimen all branching took place posterior to the inferior artery and at about the same level in the thyroid space.

failed to mention Rufus of Ephesus and Galen. The latter is accredited with being the first to record observation on aphonia and respiratory distress due to experimental paralyses produced by section or ligation of the recurrent laryngeal nerves. The next contributor of note was Vesalius[47] who in his late twenties and early thirties engaged in considerable animal experimental work in an endeavor to learn more about the role these nerves played in voice production. His drawings of the human larynx clearly depict the anatomical structures and the superior and recurrent laryngeal nerves. In general, all of the early investigators from Rufus the Ephesian to Reid[18] with perhaps the exception of Magendie[1] directed their attention to these nerves solely as they were related to phonation.

As early as 1734 Martin[19] mentioned that there prevailed a "contrariety of opinion" as to the function of the recurrent nerve. As a result of his experiments wherein the dog's voice was taken away by division of the nerves but later returned, he concluded that this return of function was due to the branches of the superior laryngeal nerve taking over the motor function that was formerly supplied by the recurrent nerve. He mentioned Galen as having stated that this return of function was due to communicating branches of the superior and inferior laryngeal nerves. The stated fact that such a communication or anastomosis exists has been and is still found in the standard text books of anatomy and otolaryngology, in spite of excellent and conclusive demonstrations by microscopic, macroscopic, and clinical evidence that negates the existence of such an anastomosis, at least as far as the motor fibers are concerned. As early as 1871 Luschka[12] stated that there was no communication between the superior and recurrent laryngeal nerves, even though they might share the same sheath for short distances. Recent experimental evidence and particularly the contributions of Lemere[25-28] leave little to be wanted in the way of conclusive proof that an anastomosis of the superior and recurrent laryngeal nerve fibers does not exist. Heighton[20] in 1792 believed that the severed nerves reunited. The probability of spontaneous reunion of the severed ends of the nerves, seems somewhat improbable.

Magendie[1] in 1813 was the first to experimentally demonstrate that the cricothyroid muscle was innervated by the superior laryngeal nerve. He gave a good description of the distribution of the recurrent nerves and believed that the muscles they supplied opened the glottis, whereas the superior laryngeal nerves, in going to the

cricothyroid and transverse muscles had exactly the opposite function: that is, closed the glottis. This teaching was blindly followed by some writers for many years. In a lecture in 1836 he stated that the anatomists were unable to solve the problem of why the recurrent nerves took such a complicated course instead of coming off directly from the pneumogastric nerves.

A brief review of the literature will impress one as to the quality and caliber of the contributors. The controversies waxed hot and several of the proponents of the more or less accepted principles and theories were unrestrained in their criticism of their opponents. The criticisms were not soley confined to the type of experiment performed and the deductions but were sufficiently broad to encompass the veracity of the investigator and his qualifications to formulate or carry on acceptable experiments. There is evident attempt by some to confine their attention to pure anatomy and others to physiology and function. None were able to confine their work to a single stated phase and consequently there was much overlapping of ideas and conclusions. Should time permit and if you are interested in the fantastic products of a fertile imagination, read an essay by Hugh Ley[21] wherein he presents his views and reasons for the upper movement of the cricothyroid cartilage during the acts of swallowing and coughing. Then read Hoover's[23] evaluation of this contribution.

One may sympathize with the student of laryngology of the early part of this century. His confusion in regard to the protean manifestations of laryngeal paralyses has been the result of the inexactness of the knowledge of the older texts such as Sajous,[37] Burnett,[35] Kyle,[36] and Bruck,[38] and feel comforted by the fact that progress ness of the knowledge of that period. One may smile smugly while reading some of the older texts such as Sajous,[37] Burnett,[35] Kyle,[36] and Bruck,[38] and feel comforted by the fact that progress has been made and that everyone should know that the internal branch of the superior laryngeal nerve is purely sensory and therefore would not be the nerve to supply all or even part of the motor filaments to innervate the arytnoideus muscle. Or do you still believe that it does? In either event, one's patronizing attitude towards the older text is due for a severe jolt when one reads the later modern texts. Ballenger[41] seventh edition, makes a positive statement that the interarytnoid muscle does receive some motor fibers from the internal branch of the superior laryngeal nerve. The com-

pletely revised 9th edition (1947) contains the same information except that it is no longer in the form of a positive statement. We now find such limitations as "possibly" and "it is thought to receive." Portmann[42] choses to ignore the minor details of the innervation of the larynx as such. In regard to the superior laryngeal nerve he makes the definite statement that this branch is exclusively sensory. On the same page, in discussing the recurrent laryngeal nerve, he states that one of the terminal branches of this nerve joins a lower branch of the upper laryngeal nerve to form Gallien's ansa. Later in the chapter, under the heading of "Paralysis" he states that total paralysis may be bilateral or unilateral: it may effect only a single muscle or all of the laryngeal muscles. We find under the caption "Paralysis of Isolated Muscles, in reality, this is more in the nature of a paresis, due to myopathies." Jackson and Jackson[39] in the 1946 edition and in those that precede it, is outstanding for obscurantism. On page 420 in regard to the internal branch of the superior laryngeal nerve we find the statement "This branch probably gives some supply to the arytenoid muscle." On page 424 we find the statement "On each side there is a superior and inferior laryngeal nerve. All four are branches of the vagi. Both pairs carry afferent and efferent impulses and all four nerves are connected by anastomotic or association fibers. In the distribution of these four nerves there is some overlapping as well as anastomoses and this is sometimes confusing in the diagnosis of paralysis." A further description of the superior laryngeal nerve: "The external (branch) chiefly motor, passes downward and is distributed to the cricothyroid muscle. A few stray neurons are traceable to an anastomisis with the inferior laryngeal nerve." On page 425 in description of the right inferior laryngeal nerve: " . . . as one nerve to the level of the upper border of the cricoid cartilage where it divides into two branches. The anterior branch is distributed to the cricothyroid, thyroarytenoid and the arytenoid muscles. The posterior branch is distributed to the right posterior cricoarytenoid muscle and to the arytenoid on both sides of the midline." In regard to the left inferior laryngeal nerve, it is stated that its position, branches and distribution are relatively the same as those of the right inferior laryngeal nerve. "In addition to the clearly defined motor and sensory innervation, the superior and inferior laryngeal nerves, there is a not so clearly understod, but definitely present, normal form of innervation known as tonus. The source of excitation impulses for this reflex tonus may be exerted through the superior laryngeal nerve

fibers or possibly, it may not be in the larynx—it may be vagal. All chains of neurons are not traceable." Such descriptions should stimulate the dullest student to a state of mild confusion and completely disorient the more brilliant ones. These specific sources of information were selected as typical of the group of accepted texts and not with the direct or ulterior motive of subjecting any of them to ridicule or undue criticism. The dissemination of an adequate explanation by King and Gregg[44] and confirmed in this paper should eliminate all opportunities for confusion.

The results of lethal injury to abductor fibers of both recurrent laryngeal nerves is well known and dramatic. Prior to the advent of thyroid surgery as an accepted procedure, there were few surgical reasons that afforded the opportunity or necessity to invade the areas of the neck through which the recurrent laryngeal nerves traverse. Associated with the popularization and vindication of the advantages of thyroidectomy in properly selected cases, came an increase in laryngeal paralyses. Involvement of the adductor fibers was and is relatively rare. The most unpleasant sequelae are aphonia or a markedly changed or impaired voice. Unilateral abductor paralysis is the most common of the postoperative complications and is seldom, if ever, annoying to either the patient or the surgeon. The quality and timbre of the voice may be unchanged and the diminution of available airway during inspiration is seldom cause for concern. The frequency of this condition is much greater than the reported figures would indicate. Goldman[50] reports on the incidence of laryngeal paresis and paralysis seen at the Thyroid Clinic of the University of California Hospital during the twelve and one half year period from July 1935 to January 1948. The results were obtained from medical appraisal and follow-up records of 1042 patients who attended the Thyroid Clinic and were operated upon for thyroid disease. From 80 to 90 percent of the operations were performed by resident staff members late in their training period, with or without operative supervision by a senior surgeon. He states "Limitation of motion or weakness of one vocal cord, indicating that the laryngeal nerve fibers were intact, was termed paresis; if, however, there was a complete abductor paralysis, the cord was said to be paralyzed. Examinations were conducted by the members of the Department of Otolaryngology." The routine surgical procedure is anatomical thyroidectomy performed by an intracapsular excision with preservation of the lateral and posterior capsule and without

visualization of the recurrent laryngeal nerves. Generally between 3 and 4 grams of thyroid tissue is left behind.

Table No. 1 is an abstraction and a selected condensation of several tables in his report. In this manner attention is focused on the incidence of laryngeal paresis and paralysis of 5.7 percent. Unfortunately he does not mention or present data referrable to the incidence of preoperative laryngeal paresis or paralysis in this group of patients.

TABLE I.

| | NUMBER | PARESIS | PARALYSIS |
|---|---|---|---|
| Toxic diffuse goiter | 379 | 3.3% | 2.7% |
| Toxic nodular goiter | 179 | 4.0% | 3.1% |
| Non-toxic nodular goiter | 389 | 2.3% | 2.3% |
| Chronic thyroiditis | 47 | 4.8% | 3.8% |
| Carcinoma of thyroid | 48 | 10.7% | 8.0% |
| Total | 1042 | 3.2% | 2.5% |

The patient with bilateral abductor paralysis presents a different picture. In the advanced cases, the difficulty with inspiration and the inspiratory howls during sleep leave no question as to the nature of their origin. In some patients these findings may be minimal and permit evading the correct diagnosis by substituting that of asthma. The amount of embarrassment to respiration extends from this minimal type to the most severe type wherein immediate tracheotomy is essential as a life saving measure.

It is evident why the surgeons doing the thyroidectomies were also avid for information in regard to the anatomic relations of the recurrent laryngeal nerves, an explanation for the varying types and extent of the paralyses, and for ways and means of avoiding these paralyses. The laryngologists were of no help. It was also evident that the mere size and consistency of the thyroid gland was not the answer. The association of excessive bleeding during surgery and the laryngeal complications was too common to be overlooked. The

next logical step was to critically review the anatomy of the area, obtain clear cut relationships between the inferior thyroid artery and the recurrent laryngeal nerve, and either devise changes in technique, which would eliminate and minimize the opportunity of injury to these nerves or present dicta which if followed would assist others to keep their percent of complications to the minimum. Woelfler[14] stated that the nerve is always anterior to the artery or branch of the artery. Kocher[15] who needs no introduction, in 1883 stated that the inferior thyroid artery always passes posterior to the nerve, comes forward on its inner side and then bends over it. Rotter[16] reported on 15 examinations. He found that only one-third followed the pattern as presented by Kocher. Strecheisen[17] presented data on the dissection of 56 bodies. His interest centered on whether or not the artery made a loop around the nerve. He made no mention as to the anterior or posterior position of the artery in relation to the nerve. Of more recent vintage are reports by Ziegelman[31] who dissected 42 recurrent and 14 superior laryngeal nerves, and Berlin[24] who reports on the dissection of both nerves in 70 cadavers, and on 72 nerves during 70 thyroidectomies. Of this latter group, 68 nerves were not identified during the operative procedure and it is presumed that they were safely concealed deep in the tracheo-esophageal sulcus. These last two contributions will be discussed in some detail.

The time spent in recounting some of the findings of the surgeons and the surgeon-anatomist is deliberate and with the intent to emphasize certain points. There is no reason to question the quality and caliber of the authors. They were interested in the problem or they would not have reported their observations. One can assume that they were open minded, observant individuals who were familiar with the fundamental technique of the dissecting room. That although their major interests may have centered about some limited area, such as whether the artery looped around the nerve or was anteroir or posterior to the artery. They could not obtain even this limited information without having exposed and isolated considerable portions of these structures. The existence of a divided nerve was established in 1832 by Meckel.[4] Yet so few instances of divided trunk nerves appear in their literature that one must conclude that the divided nerves were not seen. This could be explained on the basis that the dissectors found one or the other of the branches of a divided nerve, considered it as being the entire nerve and deposited the remaining branch or branches in the waste basket—a common reposi-

tory for important anatomy. It would appear that in the earnest endeavor to prove or disprove some particular controversial point, the investigator lost interest in the surounding and adjacent anatomy. This is understandable but unfortunate.

The article by Ziegelman[31] reports on the dissection of 23 right recurrent laryngeal nerves, 19 left recurrent laryngeal nerves and 14 superior laryngeal nerves. The stated purpose of the dissections was to establish the relations of the nerves to the thyroid arteries, thyroid gland and the inferior cornu of the thyroid cartilage. In addition 7 larynges were dissected to demonstrate the distribution of the nerves to the muscles. The illustrations are photographs of drawings which are supposedly true representations of actual dissections. They show surprising detail in certain respects but are quite lacking in fundamentals. All the recurrent laryngeal nerves illustrated are single trunk nerves, all follow a surprisingly similar course and all go to the area of the cricothyroid articulation as single trunk nerves. The illustration of the distribution of the nerve fibers to the muscles in the 7 dissected larynges is not stated to be a composite picture, yet it is too finished a product to be a diagram and too diagramatic to be a true picture. His conclusion, No. 8, states that the internal branch of the superior laryngeal nerve sends branches which communicate with the recurrent laryngeal nerve.

The article by Berlin[24] is excellent. The dissection of both nerves of the 70 cadavers was done with special attention to the relation of the nerve to the thyroid gland and the thyroid adherent zone—that area just below the anterolateral aspect of the bar of the cricoid cartilage at the level of the 1st and 2nd tracheal rings. Illustration Fig. 3d. in his article is an excellent demonstration of a divided trunk nerve. The following quotation leaves no question as to how close he was to obtaining the long sought answer. "Twice the nerve was demonstrated branching into two terminal twigs about the level of the lower pole, the anterior branch crossing through the adherent zone and ultimately joining its posterior terminal companion beneath the inferior thyrocricoid joint. One such reasonably sized anterior branch was accidently cut at operation on the left side, after first identifying the posterior twig which was thought to be the main stem of the nerve, with resultant partial temporay paralysis of the corresponding vocal cord." Since it was the anterior branch that was cut, the paralysis should have been of the adductor type. One wishes that a detailed description of the larynx was available.

One other anatomic relationship has been cause for considerable controversy. Are there occasions when the recurrent laryngeal nerve penetrates and courses through the thyroid gland substance? The answer to that question is unqualifiedly that the nerve does not transgress the gland parenchyma. In those instances wherein the thyroid gland is markedly enlarged and the posterior surface is nodular, the nerve, lying in one of the sulci may appear to transverse the gland substance. Careful dissection and microscopic sections will show that each has retained its integrity. Embryologically it is impossible for the nerve to do otherwise.

Discussions and arguments on several points of mutual interest with Dr. Brien T. King resulted in agreement that there must be a simple, logical and rational answer to the bizarre types of laryngeal paralyses. When the answer was available it should not consist of complicated arrangements of nerve fibers which violate the fundamentals of nerve fiber distribution or necessitate establishing intricate pathways for nerve impulses that do not exist in other parts of the body. It was evident that the explanation must be found in the areas adjacent to the larynx and trachea.

Late in 1947 King was convinced that there was a simple answer, that the nerve did divide extra-laryngeally into trunks of abductor and adductor fibers and that there was experimental evidence to prove this.[27] The few dissections he had done following the realization of the importance of divided trunk nerves showed the presence of such divisions in the danger area. King and Gregg[44] reported their results in 1948. More dissections were necessary in order to verify these findings.

Since the information to be obtained was of equal if not more practical value to the thyroid surgeons, I called on Dr. William H. Rustad (of the Division of Surgery of the University of California School of Medicine, San Francisco, California) for collaberation. It is only because of his dogged perseverance in pursuing available cadaver material, and in his complete disregard for the sacrifices of his time and efforts that this material is available. Many more than 100 cadavers were dissected. The courses of the recurrent nerve and inferior thyroid artery were charted on specially prepared forms. In this manner, the interrelationships of these two structures and the basic anatomy of the area were preserved in graphic form. These charts were supplemented, when necessary, with explanatory notes.

They are available for study or review at any time. In retrospect I would add one further step for completeness—a photograph of each dissection.

Sufficient dissections were done to permit the selection of 100 cadavers wherein dissections of both the right and the left nerves and inferior thyroid arteries was possible. This eliminates a preponderance of right or left sides, and permits comparison of the two sides of each specimen. Material, which for one reason or another, had had the nerve or artery distribution of one side destroyed or invaded was eliminated. The accompanying drawings and tables are self-explanatory. No attempt was made to follow the branches of the nerves to their terminal distributions. The average available cadaver material does not permit this to be done as a simple gross dissection. The nerve branches soon become minute threads that are indistinguishable from strands of connective tissue. With little effort, and a minimum of wishful thinking, one can produce a complicated network of pathways and anastomosies which would confuse anyone. Grossly, this artifact network appears real, but would not stand up under magnification by either the dissecting microscope or microscopic sections. A few attempts to accurately follow these intralaryngeal branches was sufficient to demonstrate the difficulties that would be encountered. A realization of these difficulties engendered a more understanding attitude toward those who, in all good faith, produced complex nerve anastomoses and reported what they saw as factual evidence.

## COMMENT

It would be incomplete to offer this material without giving credit to Brien T. King, M.D. of Seattle, Washington, for his two contributions to laryngology. The number one contribution was the presentation[43] of the first satisfactory reconstruction operation for rehabilitation of individuals with bilateral abductor paralysis. The second contribution[44] was the recognition that the recurrent laryngeal nerve does divide into abductor and adductor trunks in the extralaryngeal space. His findings that these divisions into two branches in the extralaryngeal space are not an anatomical rarity but occur sufficiently often to constitute an accepted deviation from the routine text book description, namely, that the recurrent laryngeal nerve is a single trunk nerve. In this published article he presented

the evidence to account for the behavior of the adductor and abductor groups of muscles. This contribution nearly solved the problems that have been the basis for years of controversy.

The initial scope of the present work was essentially to confirm or deny the statement that the recurrent laryngeal nerve was found as a divided nerve trunk in the extralaryngeal areas in a appreciable percent; that is, sufficient to be of practical importance. In other words, the possibility of the nerve dividing in this area, prior to entering the larynx, was no discovery. It had been reported as a fact by Meckel[4] and others, but was considered as an anomaly primarily confined to the right side of the neck. That there could be any relationship between this extralaryngeal division of the nerve, and the accepted knowledge that the recurrent laryngeal nerve normally divided into an abductor and adductor branch immediately after it passed behind the cricothyroid articulation, was completely ignored by both the anatomists and the more erudite clinicians. King and Gregg[44] pointed out the significance. If the recurrent laryngeal nerve did divide in any percentage approximating the 30% reported by Dr. King, it was essential that additional dissections be done in order that statistical variations or errors be kept to a minimum. The basis for his statistics (16 cadavers or 32 nerves) was not sufficient.

The results obtained from the reported 100 cadavers did confirm the fact that the recurrent laryngeal nerve does divide in the extralaryngeal space. The percent incidence was substantiated to a surprising degree. This confirmation of the figures is important. Of more importance was the finding of secondary and tertiary branches of either or both the abductor or adductor trunks in the extralaryngeal space. The area of these further divisions was confined to the superior thyroid and thyroid areas. The dissections, as they were developed, did not show secondary or tertiary branches lower than the thyroid area (Figs. 3 and 4). The demonstration of these divisions of the divided nerve trunks offers a complete explanation of the so-termed bizarre or controversial types of laryngeal paralysis. For the first time, one can readily understand the production of laryngeal paralysis that is confined to a single muscle or a small group of muscles. The explanation rests on the possibility of injuring or destroying one or more of these smaller branches. Likewise, we have for the first time a logical explanation of the

origin of changes in quality or timbre of voice or both, and of voice fatigue following surgical trauma. These changes are subjectively evident but are difficult if not impossible to demonstrate by direct or indirect laryngoscopy. Injury to one or some of these terminal branches is the logical answer. The demonstration of these smaller branches in the vulnerable area is sufficient to conclude that they can be injured. We are therefore equipped with sufficient anatomical material to explain any and all of the types of laryngeal paralysis and paresis that follow thyroid or other surgery in this area. The demonstration of the initial divisions of the recurrent laryngeal nerve into adductor and abductor trunks in the extralaryngeal area was a great contribution. However, it limited the explanation of the laryngeal paralyses to a total adductor or abductor type. It did not permit a logical explanation of partial adductor or abductor involvement or partial combinations of both nor could the simple separation of the two types of fibers explain a single muscle paralysis.

There are obvious reasons why there is little if any opportunity to carry out clinical experiments on the normal living human subject. There are reasons to question the validity of the findings produced by stimulation of the nerve branches or main adductor and abductor trunks or either of them in the deeply anesthetized patient or in the muscle responses in the larynx that is grossly infiltrated by malignant neoplasm. In a few personal experiences it was evident that a larynx that was sufficiently infiltrated with neoplasm to show fixation of the vocal cord on attempted voluntary motion showed no additional motion on artificial stimulation. The opportunities to obtain reliable results from stimulation of the major trunks or their branches in the living human subject are extremely limited.

Murtagh and Campbell[46] offer the thesis that there is a difference in size between nerve fibers of the adductor and those of the abductor nerves; the latter being predominantly the smaller and the less myelinated of the two. Now that attention has been directed to the fact that the recurrent laryngeal nerve is found as a divided trunk in the extralaryngeal space sufficiently often to eliminate this condition as being a rarity it should be an interesting and worthwhile project to obtain from fresh cadavers specimens of these branched nerves, and subject these divisions to microscopic examination. A rough protocol for correlating the microscopic and gross anatomic findings is intriguing. If the conclusive experimental and clinical

work demonstrating that the initial division of the recurrent laryngeal nerve separates the adductor and abductor fibers is substantiated by the microscopic sections, the cause for controversy will be settled for all time. If such an investigation is not under way, it should be started. The material for study of the normal nerve fiber distribution is easily available. The material from known cases of laryngeal paralysis of peripheral origin is limited. Even so, it should require only a few selected cases to complete a well rounded protocol.

In a preliminary report we[49] presented this material with attention directed to the fact that the finding of a divided recurrent laryngeal nerve was not an anatomical anomaly. It was emphasized that the surgeon invading the thyroid area should expect to find one or both nerves as divided structures in 43% of the patients. We emphasize this point to discourage the false sense of security that accompanies the exposure and isolation of a sizeable nerve in the danger area. It is no longer acceptable to consider any identified nerve in this area as being the entire recurrent laryngeal nerve unless one proves it to be so by following it to at least the level of the clavicle. This is not feasible as a routine procedure. The finding of secondary and tertiary branches in the extralaryngeal area and the ultimate and variable nerve-artery relationships were stressed to encourage the surgeon to bypass the unpleasant sequelae of laryngeal paralysis by avoiding mass clamping and ligature or suture to obtain hemostasis. The knowledge of the branched nerve and the inconstant relation of the nerve and its branches to surrounding anatomical landmarks, as well as the major branches of the inferior thyroid artery, leaves no opportunity for any single surgical approach to eliminate all the opportunities or probabilities of nerve damage. The knowledge of the variations in anatomical relationships in the complex nerve-artery associations does equip the surgeon to proceed in this area with the chances of nerve damage at the level of the irreducible minimum. These statements are made with the full realization that in spite of this knowledge and meticulous surgical technique, there will be instances wherein nerve damage cannot be avoided. Varying percentages of nerve damage following thyroid surgery are available.[22, 50]

Frankly, the significance of the finding of secondary and tertiary branches in the extralaryngeal space was not realized until the total number of dissections was nearly complete. The initial objective of the dissections was to demonstrate the presence or absence of divided

nerve trunks and to note the area where the branching occurred in relation to the thyroid gland and the inferior thyroid artery. A review of the completed chart diagrams of the dissections to determine the relative positions of the nerve and the major branches of the inferior thyroid artery showed the presence of the secondary and tertiary divisions. Also evident was the opportunity to incorporate some one of the branches of a multi-branched nerve in a ligature or suture. The recognition of this fact supplied the missing information essential to satisfactorily explain the less common and less extensive types of laryngeal paralysis.

## CONCLUSIONS

1. The dissection of 100 cadavers (200 recurrent nerves) shows conclusively that the division of the recurrent laryngeal nerve into two or more branches in the extralaryngeal area is not an anatomical rarity.

2. The variable and complex nerve-artery relationship establishes a potential nerve-damage-hazard associated with all surgery in the thyroid area.

3. One should expect to find divided trunks in 43% of adult humans.

4. Injury to one or the other trunks of a simple divided nerve will produce a pure adductor or abductor paralysis.

5. It was demonstrated that secondary and tertiary divisions of the recurrent laryngeal nerve occur in the extra laryngeal area.

6. The presence of these secondary and tertiary nerve divisions in this vulnerable area offers an adequate explanation for the more complex and heretofore controversial types of laryngeal paralyses.

7. Injury to one branch of a multi-branched nerve will produce paralysis only in the area supplied by that branch. In some instances, only a single muscle or a portion of a muscle may be supplied by this branch.

8. The present explanation of the various laryngeal paralyses following surgical trauma eliminates the necessity for fabrication of motor filaments in the otherwise purely sensory internal branch of the superior laryngeal nerve.

9. There is now available an adequate and logical explanation for all of the types of laryngeal paralyses found following surgery in the thyroid area. The explanation is based entirely on injury to the recurrent laryngeal nerve or its branches sustained during the surgical procedure.

TABLE II.

| | | |
|---|---|---|
| Bilateral Single Trunk Nerves | (70) | 35% |
| Bilateral Divided Trunk Nerves | (48) | 24% |
| Mixed Single and Divided Trunk Nerves | (82) | 41% |

TABLE III.

LOCATION OF DIVISIONS OF NERVES (86 NERVES)

| | | |
|---|---|---|
| Superior Thyroid Space | 21 | 24.2% |
| Thyroid Space | 47 | 54.7% |
| Inferior Thyroid Space | 18 | 20.9% |

TABLE IV

RELATION TO BRANCHES OF THE INFERIOR THYROID ARTERY (200 NERVES)

| | ANTERIOR | BETWEEN | POSTERIOR | PARALLEL |
|---|---|---|---|---|
| Right Single Trunk Nerves | 19 | 9 | 18 | 10 |
| Right Divided Trunk Nerves | 17 | 17 | 20 | 8 |
| Left Single Trunk Nerves | 14 | 12 | 33 | 12 |
| Left Divided Trunk Nerves | 5 | 14 | 22 | 4 |
| Total | 55 | 52 | 93 | 34 |
| Percent | 27.5% | 26.0% | 46.5% | 17.0% |

TABLE V.

NUMBER OF DIVISIONS OF DIVIDED NERVES

86 NERVES)

| | 2 | 3 | 4 | 5 | 6 |
|---|---|---|---|---|---|
| Right | 40 | 4 | 6 | 1 | |
| Left | 27 | 5 | 1 | 1 | 1 |
| Total | 67 | 9 | 7 | 2 | 1 |
| Percent | 77.91% | 10.46% | 8.14% | 2.32% | 1.16% |

## CHRONOLOGICAL DATA

100 A.D. Rufus The Ephesian. Greek anatomist. Treatise on Nomenclature on Parts of the Human Body. Aphonia and respiratory distress associated with injury of the recurrent laryngeal nerves.

160-200 A.D. Galen. The first recorded observations on experimental paralysis of the recurrent laryngeal nerves. Animals . . . aphonia and respiratory distress.

1514-1564. Vesalius.[47] Interested in the role the recurrent laryngeal nerves played in voice production. Experimental work and drawings of the human larynx showing the structures and the superior and recurrent laryngeal nerves.

1734. Martin.[19] Animal experiments. Return of voice (dog) due to communicating branches of the superior and recurrent laryngeal nerves.

1792. Haighton.[20] Believed that the severed ends reunited.

1813. Magendie.[1] First to demonstrate experimentally that the cricothyroideus was innervated by the superior laryngeal nerve.

1823. Stedman.[2] Anomalous right recurrent nerve. Explanation was not adequate.

1826. Hart.[3] Irregular origin of the right recurrent laryngeal nerve. Secret of the circuitous route was to be found in the natural law of embryonic development.

1828. Rainey.[8] Anomalous right recurrent laryngeal nerve.

1832. Meckel.[4] First to make mention of a divided recurrent laryngeal nerve. Rare but when it did occur it is always on the right side.

1836. Ley.[21] Fertile imagination as to the function of the cricoid cartilage.

1836. Magendie.[1] Anatomists unable to explain why the recurrent laryngeal nerves took such a complicated course.

1837. Hilton.[9] Anomalous right recurrent laryngeal nerve.

1838. Reid.[6, 18] One of the first to present experimental studies that approached completeness and were of scientific value.

1846. Hérard.[5] Anomalous right recurrent nerve.

1847. Reid.[6] Gave correct explanation of the origin of the anomalous recurrent nerves.

1848. DeMarquay.[29-quoted] Anomalous recurrent laryngeal nerve.

1869. Philipeaux and Vulpian.[11] Anastomosing fibers when present come from the superior laryngeal nerve.

1871. Luschka.[12] No communication between the superior and recurrent laryngeal nerves even though they might share the same sheath for short distances.

1876. Habershon.[10] Communications between the superior and recurrent laryngeal nerves.

1879. Woelfler.[14] Dissections. Nerve always anterior to the artery.

1881. Semon.[32-quoted] Formulated and presented his Law.

1883. Brenner.[7] Anomalous right recurrent laryngeal nerve.

1883. Kocher.[15] Dissections. Artery posterior to the nerve.

1884. Exner.[13] Elaborate monograph.

1885. Rotter.[16] Dissections (fifteen). Only one-third followed the pattern described by Kocher.

1886. Streckeisen.[17] Dissections, 56. Mainly interested as to whether or not the artery formed a loop around the nerve.

1887. Hooper.[45] Excellent article . . . both historically and practically.

1892. Russell.[32] Substantiated Semon.

1906. Grossmann.[33] In certain cases of paralysis the cords assume the cadaveric position due to the atrophy of disuse of the cricothyroid muscle. Work has not been confirmed.

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4. Meckel: Manuel of Anatomy (translated by Doane), 1832.

5. Herard: Bull. de la Soc. anat. de Paris, p. 111, 1846.

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LII

# THE PLACE OF TRACHEOTOMY IN HEAD INJURIES

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Normal brain tissue is susceptible to damage from anoxia, and the brain injured, whether it be as a result of trauma, operation, inflammatory processes, impaired circulation, or space occupying lesion is more susceptible to anoxia.

It is felt that anoxia can be prevented in many cases of cerebral trauma by means of a tracheotomy where the simpler methods for releif have failed. In 1859 Marshall Hall stated in the Lancet that we must regard tracheotomy as a heroic remedy only appropriate to herculean forms of disease. Today in expert hands it is a dramatic but harmless procedure carrying no additional risk for any patient with secretory obstruction in the tracheo-bronchial tree.

Galloway[1] was one of the first to show the value of tracheotomy in combating cyanosis and flooding of the lower respiratory tract. The flooding of the lower respiratory tract in comatose patients with brain injury is due to the failure of the cough mechanism which normally clears the respiratory passages of accumulated secretions. This cough mechanism consists of inspiration, closure of the glottis, increase in intrathoracic pressure, opening of the glottis and sudden expiration with release of the intrathoracic pressure. Any interruption in the physiology of this mechanism by coma or depression of the respiratory centre in the medulla will retard or abolish the cough reflex.

Respiratory obstruction causes an elevation of the intrathoracic venous pressure due to the laboured breathing necessary to overcome the partial obstruction which results in a reduction of the ventilating capacity of the lungs— any increased intrathoracic pressure is readily transmitted to the intracranial venous system producing venous congestion of the brain resulting in increased intracranial pressure which in turn produces anoxia.

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Practically every patient with a severe cerebral trauma is in danger of respiratory obstruction from salivary, pharyngeal and tracheo bronchial secretions. Any attempt to swallow may result in entry into the larynx of fluids and solids. If vomiting occurs there is a grave danger that the vomitus may be inhaled and respiratory infection follow. Many of these patients appear to salivate profusely, and the presence of a feeding tube often causes irritation which increases the accumulation of secretions in the pharynx. The respiratory passages are not cleared of the accumulated secretions due to the ineffectual cough so that stagnation of the secretions occurs necessitating constant medical attention to prevent atelectasis and infection. Other cases may become obstructed due to a relaxed pharyngeal musculature, laryngeal spasm, paralysis of the vocal chords or accumulation of dried exudate on the chords or subglottic area, and in children following prolonged endotracheal anaesthesia subglottic oedema is not infrequent. In critically injured brain cases decerebrate rigidity may be present resulting in the inability to clear the tracheobronchial secretions.

Partial respiratory obstruction also causes hypoventilation so that the ratio between the amount of air breathed in per minute and the amount of oxygen consumed per minutes falls; as hypoventilation becomes more severe carbon dioxide is retained in the body with an increase in the carbon dioxide content of the systemic and cerebral arterial blood which causes a marked dilatation of the cerebral vessels. Hypoventilation creates a fall in the oxygen content of the arterial blood bringing about an arterial anoxemia. When the cerebral vascular endothelium becomes damaged by anoxemia, it is permeable to fluids, resulting in edema, and haemorrhage is apt to occur. This arterial blood of low oxygen content is also a dilator of cerebral vessels.

The venous congestion and the arterial dilatation raises the intracranial pressure and bleeding from the cerebral vessels may occur. The intra-cranial pressure which in time depresses the medullary centre, accentuates the inadequate airway and decreases the aeration of the lungs thus establishing a vicious circle.

## REPORT OF CASES

CASE 1.—S. H. A 70 year old female admitted to Toronto General Hospital in deep coma following an automobile accident. There

was no external sign of head injury, there was no response to painful stimuli and the respirations was 30-40 per minute. A few hours after admission extreme muscular effort became associated with breathing and a tracheotomy was performed.

There was considerable improvement in the ariway but the patient died three days later from midbrain and cerebral contusions. The temporary improvement indicated that the anoxia was partly responsible for the loss of consciousness.

CASE 2.—E. B. A 57 year old female admitted to hospital because of fatigue and inability to carry on her usual household duties. For the past two weeks previous to admission she complained of headache in the frontal region more particularly on the right side and which was associated with nausea and for the past two weeks was "befuddled" mentally and had occasional incontinence of bladder and bowels. Little to find on physical examination other than the patient was drowsy and would fall off to sleep during examination. There was an early papilledema. Pulse Slow 50-60 per minute. Ventriculogram showed a large space occupying lesion in right frontal lobe. A resection of the right frontal lobe was carried out removing a cystic astrocyloma.

The patient never regained consciousness following operation and twenty hours later developed acute dyspnea. A tracheotomy was performed but the patient died twenty hours later.

Autopsy was not obtained but in all likelihood the patient had involvement of the brain stem from uncal herniation at the time of operation.

CASE 3.—G. N. A 47 year old man admitted to hospital 45 minutes after having been struck by a car. He was well orientated as to time, place and person. Two hours later he gradually became confused and lapsed into deep coma with stertorous breathing. The left pupil was larger than the right and fixed, a spinal tap showed gross blood.

He showed decerebrate rigidity and the temperature rose to 105.° Following a removal of a subdural haematoma there was increased respiratory difficulty and more generalized muscular hypertonia. A tracheotomy was performed followed by marked improve-

ment in the patient's general condition. Subsequently the patient was at a low level unconsciousness and died six weeks following injury.

CASE 4.—B. U. A 17 year old boy sustained a compound fracture of the right frontal region, was hospitalized three weeks, returned to work two and a half months following his injury. Six months later was admitted to Toronto General Hospital because of headaches gradually becoming more severe and were frequently associated with nausea and vomiting. He also lacked ambition and energy and two weeks prior to admission began to have difficulty with his speech. On admission he lay in a position of opisthotonus, breathing rapidly, temperature 103.° He was confused and disorientated, early papilledema was present on the right side and left lower facial weakness was present. Ventriculogram revealed a space occupying lesion in the lower portion of the right frontal lobe. Operation revealed a large right frontal lobe abscess.

Following operation, cough was ineffectual and patient had accumulation of tracheobronchial secretions. Tracheotomy performed through which large amount of secretions were aspirated. The patient did not improve very much, though the spinal fluid cleared. He did not regain consciousness following operation, he became quadraplegic and paralysis of his respiratory muscles took place.

CASE 5.—H. P. An 18 year old boy struck by a car while riding a motor scooter and rendered immediately unconscious. On admission to hospital he was deeply comatose. Skull films did not show any gross evidence of fracture. Twenty-four hours after admission his temperature rose to 104° and respirations between 50 and 60 per minute—cough ineffectual. Tracheotomy performed and progressive improvement followed establishment of an adequate airway. Due to the depressed conditions there was an absence of cough reflex permitting accumulation of secretions. Patient discharged two weeks following admission.

CASE 6.—L. S. A 51 year old man was admitted to hospital giving history of good health until 10 days previous to admission when he became drowsy, mentally confused, some speech difficulty and unsteadiness in walking. Ventriculogram indicated tumour in the left frontal lobe. A left frontal lobectomy was performed for a

malignant tumour. Following operation the patient became cyanosed, had considerable difficulty with his breathing and anoxic, before a tracheotomy was carried out.

There was marked improvement in cyanosis and airway following tracheotomy but patient had a slow and unsatisfactory recovery. He was discharged from hospital six weeks after admission being aphasic though he could understand what was said to him. He did not have any motor paralysis.

If the tracheotomy had been more quickly performed it is unlikely this patient would have had any permanent damage to the central nervous system from the anoxia.

CASE 7.—L. H. A 56 year old man was admitted to hospital with a gross arterio venous malformation on the left side of the neck involving the internal maxillary and superficial temporal artery and alsa a small aneurysm of the right internal carotid. He also had moderate cardiac decompensation secondary to the arteriovenous aneurysm.

Two attempts to stab the right common carotid for an arteriogram failed and a cut down was done under general anaesthesia (pentothal). Following the closure of the wound the patient began to struggle and while he was being lifted on to a stretcher he coughed out his endotracheal tube. On the way back to his ward he became very cyanosed and stopped breathing. After artificial respiration and coramine his heart began beating and respirations resumed but were ineffectual. The endotracheal tube could not be re-inserted and a tracheotomy was carried out with the resumption of normal respirations and colour.

LARYNGOSPASM FOLLOWING PENTOTHAL AS AN ANESTHETIC

CASE 8.—D. N. A 14 year old boy fell 30 feet from a tree to the ground becoming immediately unconscious. Eight days later admitted to Toronto General Hospital semi comatose, more or less restless, was never conscious enough to talk. The right pupil was dilated and fixed, the right ear drum perforated. Complete right hemi-paresis. Temperature 104.° Following upper mid temporal burr holes for subdural hematoma, the patient developed respiratory obstruction due to accumulated secretions so that a tracheotomy was

performed. The airway was greatly improved and the tracheotomy tube was removed in three weeks. After remaining semi-comatose for nearly two months the patient gradually began to respond and on discharge from hospital, was alert, attentive, interested in his surroundings and orientated in all spheres.

CASE 9.—R. S. A 23 year old man admitted to Toronto General Hospital eight hours after having been struck by a car while riding a bicycle. He was a known epipleptic from early childhood. On admission he was deeply comatose, with blood and brain tissue escaping from left ear. X-rays revealed multiple skull fractures, short rapid respirations suggested some brain stem drainage, all four limbs were hypertonic. Following a second temporal decompression the patient had some respiratory difficulty with dramatic improvement following tracheotomy.

This patient was discharged from hospital 5 months after admission having slowly but gradually improved in regard to his level of unconsciousness. On discharge from hospital he was reasonably well orientated though attention was poorly sustained. It was felt that tracheotomy saved this young man's life.

CASE 10.—M. S. A 14 year old girl was thrown from a horse, eight hours later was admitted to Toronto General Hospital deeply comatose and showed signs of rigidity. Following removal of a subdural hematoma, her respirations became more labored and she was becoming continually plugged with mucus. A tracheotomy wsa performed with marked improvement of her respiratory problem. He state of unconsciousness did not change and she was sent home with a nurse five weeks after admission with the tracheotomy tube in place.

CASE 11.—A. T. A 68 year old man, eight hours before hospital admission complained of sudden severe headache in vertex and within a few moments was unconscious. A large subdural hematoma removed. Four weeks following operation, the patient's condition was precarious and due to ineffectual cough he had an accumulation of tracheobronchial secretions. A tracheotomy was carried out with marked respiratory improvement. Discharged from hospital 6 weeks after admission with almost full bladder control and active movement of right upper and lower limbs which were almost equal to those of the left.

It was felt that tracheotomy saved this patient's life when the general condition was very precarious.

## COMMENT

Eleven cases are reported in tracheotomies well performed after severe brain injuries. Four of these patients died most likely because of midbrain and cerebral contusions or the presence of extensive damage to the medullary respiratory centre which may explain failure of a tracheotomy to restore normal breathing. In some cases it may be difficult to interpret whether or not the pulmonary oedema is of cardiac origin in which case tracheotomy is of no avail. The average age of the group that died was 47 years and that of the group which survived was 35 years. It was felt that this later group of patients would have died if tracheotomy had not been carried out.

Many of the usual procedures such as postural drainage, hypoventilation by means of blow bottles or carbon dioxide inhalations and bronchoscopy are impractical or dangerous to patients with brain injuries. Aspiration through an endotracheal tube should only be carried out as an emergency measure to maintain a patent airway during the performance of a tracheotomy, as it may cause trauma and further laryngeal surgery will be necessary if left for any length of time. Bronchoscopic suction is ideal for removal of isolated plugs of mucus or in mild cases of secretory obstruction, but in many cases it is contraindicated especially in head injury cases complicated by fractures of the cervical vertebrae.

## CONCLUSION

It should be stressed, where the simpler procedures for the removal of the tracheo bronchial secretions have failed, that a tracheotomy should be performed in all patients with cerebral injury who are comatose or semi-comatose and show signs of respiratory distress or cyanosis.

The indications for tracheotomy in brain injury vary in individual cases, and the indications are not necessarily those of the classical picture of dyspnea due to laryngeal obstruction. Other indications for the procedure of tracheotomy are patients with relaxed pharyngeal musculature, loss of swallowing and gag reflexes, crusting in the pharynx and larynx which leads to exhaustion of the patient due to the muscular effort associated with the labored breathing

required to overcome the partial respiratory obstruction. One should not forget that the presence of a feeding tube increases the amount of pharyngeal secretions and may cause laryngeal inflammation or perichondritis. Finally unconscious patients are often very sensitive to slight degrees of oxygen want and even minor degrees of cyanosis are important as it may not be recognized due to loss of blood or shock.

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# Society Proceedings

## CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY

*Meeting of Monday, December 3, 1951*

THE PRESIDENT, DR. ARTHUR J. COOMBS, IN THE CHAIR

### Intralaryngeal Arytenoidectomy for Bilateral Abductor Vocal Cord Paralysis

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(Abstract)

The intralaryngeal approach for the surgical treatment of bilateral abductor vocal cord paralysis is presented. Arytenoidectomy from within the larynx while using the Lynch suspension apparatus for visualization of the larynx has been found feasible and not particularly difficult for one trained in the use of the suspension apparatus. For those surgeons without such training and equipment, the King operation or one of its modifications will and should continue to be the operation of choice.

A few minor modifications have been added to the technic of intralaryngeal arytenoidectomy as presented previously. The removal of the arytenoid cartilage by means of the intralaryngeal approach is carried out with the larynx exposed to direct inspection through the mouth by means of the Lynch suspension apparatus. If the patient is not already wearing a tracheotomy tube, the first step in the procedure is to insert one. This may be done either at the same time as or a few days prior to the arytenoidectomy. Following tracheotomy, the oropharynx, hypopharynx and interior of the larynx are anesthetized by topical application of a fresh solution of 5% or 10% cocaine. General anesthesia is then induced by the use of curare and pentothal sodium administered intravenously, after which the suspension laryngoscope is placed in position. Topical applications of cocaine are next made to the vocal cord and the aryepiglottic fold. An injection of procaine with adrenalin or adrenalin alone may be made into the area to minimize bleeding. An incision

one centimeter in length is made over the superior surface of the arytenoid cartilage approximately one to two millimeters medial to the cartilages of Santorini and Wrisberg so as to avoid these cartilages in the dissection. The primary incision is carried deep into the tissues down into the arytenoid cartilage. The incision extends anterolaterally into the aryepiglottic fold. By using a suitable grasping forceps, the superior border of the cartilage is identified. Dissection of the arytenoid cartilage is then carried out. The various muscular attachments are separated from the cartilage in its lateral aspect. The superior portion of the cartilage is then rotated laterally so as to facilitate separation of the muscular attachments on the medial aspect of the cartilage. The cartilage is then removed. A very sharp laryngeal knife, sharp spatula or scissors is used to carry out the dissection. It is important to avoid tearing or injuring the mucous membrane on the lateral wall of the larynx because scarring in this area may nullify the attempt to increase the glottic opening.

Following complete removal of the arytenoid cartilage, electrocautery may be employed into the depths of the wound and lateral to the vocal cord. The tissues within the wound are lightly cauterized on the lateral aspect of the wound only. Lateral fixation of the posterior two-thirds of the cord may be made more certain by the scarring which follows the use of the electrocautery. Many cases have been operated upon in which electrocautery has not been used and the end results have been successful. At the end of the operation, an acrylic obturator may be introduced between the cords to be left in place for a period of three to seven days. The tracheal cannula may be removed after three to six weeks.

With the various improved technics offered, the operating time in performing the intralaryngeal arytenoidectomy has been considerably reduced. This procedure is attended by very little postoperative reaction and the end results have been uniformly successful in the author's nine cases as well as in 61 additional cases reported to him by other surgeons. In four of the 70, it was necessary to perform an arytenoidectomy on the opposite side because of an inadequate airway. Excellent results were obtained in three of these patients.

A motion picture was presented showing the technic of suspension laryngoscopy. Various diagrams were shown demonstrating the technic of the procedure. Recordings of the voice taken before and after surgery were presented.

DISCUSSION

DR. SAMUEL J. PEARLMAN: I think we all agree that this was a stimulating and interesting presentation. Two thoughts may be

permitted, namely, the effect of bilateral abductor paralysis on the voice and, more important, on the life of the patient. Up to the time of King efforts at cure were sporadic, on occasion based on faulty notions and inadequate from a technical viewpoint. To King, therefore belongs credit for initiating successful treatment of a condition which must be faced so long as surgical care for certain thyroid conditions remains the treatment of choice.

It is of interest to dwell upon the two chief approaches toward cure. In one the emphasis was on an approach to the arytenoid cartilage from without, avoiding the interior of the larynx and so rotating the cartilage as to produce the desired result. It must always be a fine point to determine how much airway the patient must be given, for the better the airway the worse the voice.

The operations designed to keep the arytenoid cartilage properly rotating outward must remain, under present circumstances, difficult, and are the prerogative of a few gifted surgeons who have enough material available to become highly qualified. As one reads of the experience of these men, the difficulties became apparent. The attempt to sew the arytenoid in the proper place may tear or break the cartilage and efforts to expose it properly may, according to most observers, result in a tear into the interior of the larynx, with granuloma formation and a scar which vitiates a possible good result. To escape this difficult project of outwardly rotating the cord and, at the same time, preserving the arytenoid cartilage, there are those who proceed at once to routine removal of the cartilage. It is claimed that healing and scar contracture will, with proper care, usually find the cord in the desired position. As a proponent of this approach Dr. Thornell deserves great credit, and his results appear to be as good as any. His approach has the virtue that it is simple if one has the skill necessary to do suspension laryngoscopy.

There are one or two points upon which I hope he will speak. Is it really so that it jeopardizes the good result if the interior of the larynx is invaded, or have we feared that contingency too much? Dr. Thornell warns against tearing into the larynx, but his method is an intralaryngeal one. Does he mean that we should avoid undue trauma? The fact remains that intralaryngeal approaches do give good results.

There are members here tonight who have had a large experience similar to that of Dr. Thornell, and I hope they will share the discussion. As long as these cases are here, and it appears that they will be for a long time, this approach and solution seem to be a long step in the direction of complete success.

DR. PAUL HOLINGER: The problem of bilateral recurrent laryngeal nerve paralysis is one that Dr. Thornell has returned to endoscopic laryngology in a very dramatic way. I should like to congratulate him on his ingenuity and thank him for this presentation. In the past five years we have seen 72 cases of bilateral abductor nerve paralysis, 64 of which followed thyroidectomy and, of these, we have used the intralaryngeal procedure in 15 cases. Therefore, we add to Dr. Thornell's reported cases an additional series which further demonstrates the effectiveness of the procedure described. We are convinced that it is practical and gives good results. That does not mean that it replaces other procedures or that we consider other procedures antiquated. Selection of an operative technic is up to the surgeon; one may obtain better results with his operation and another with a different procedure, yet the end results may be the same in the two series.

One interesting feature of abductor paralysis which we have found important has not been mentioned this evening; that is, the medical management of the patient. We have found that almost every one of our patients has had hypothyroidism and many of them hypoparathyroidism, because when the surgeon severed the nerves he had resected too much thyroid tissue and, in some cases, had also removed the parathyroids. A high cholesterol and low blood count are much more significant than a normal or even elevated metabolism rate. If the patient's thyroid activity is controlled first, his breathing is easier. If it is kept under control after surgery, he has a better opportunity to maintain a good airway. It is important to determine his parathyroid activity by obtaining serum calcium levels; not infrequently these patients have respiratory spasms that are corrected by adequate calcium administration. Dr. Keeton and Dr. Ranke have examined our patients carefully on these points, and it is surprising to see how many were myxedematous in spite of the fact that the basal metabolic rate was normal.

We feel that the use of the plastic obturator is very important, and it is used in our cases. We had the good fortune to have Dr. Thornell help with one of these procedures and we found, as he suggests, that the plastic mold is of great help.

Dr. Tornell mentioned that the incidence of bilateral paralysis is not related to the skill of the surgeon. I wonder if the use of intratracheal anesthesia may, in part, be responsible for the increased incidence. The surgeon who does a thyroidectomy without an intratracheal airway is aware that something has happened if one nerve is cut. If an intratracheal tube is used, the patient has no difficulty

whatsoever in breathing until the tube is removed. It would seem that the use of intratracheal anesthesia in thyroidectomy adds a certain risk, although it removes what is probably a greater hazard in the majority of cases, namely, respiratory obstruction. Probably with increasing awareness of the tragedy of bilateral paralysis the surgeon may avoid this complication, the incidence of which is increasing so rapidly.

DR. HANS VON LEDEN: I would like to commend Dr. Thornell on his presentation. Several years ago at a meeting of the Academy I had a preview of this operation in the form of a preserved arytenoid cartilage, which I think he had removed from his first case. I was impressed at this meticulous dissection, and the skill and surgical technic used in the procedure.

In discussing this subject, it is important to evaluate the difference between the extralaryngeal and intralaryngeal approach by minimizing the imponderables over which the surgeon has no control. As Dr. Pearlman mentioned, and as Dr. Kelly has pointed out with reference to his operation, there are many imponderables in the extralaryngeal approach: the difficult exposure of the arytenoid cartilage by any external approach; the suture material which may break or cut through the cartilage; the formation of granulomata due to mucosal lacerations; and the occasional necrosis of cartilage. Any intralaryngeal operation that eliminates these imponderables over which the surgeon has no control is certainly preferable to the extralaryngeal procedure.

I would like to stress the use of the electric cautery as mentioned by Dr. Thornell. It is interesting to watch the cord draw aside as the cautery is applied. For me, at any rate, it is a great deal easier to watch the cord moving aside and then stop at the point which I think provides an adequate airway rather than to rely on someone else looking into the endoscope while I maneuver the suture.

My second point refers to intralaryngeal arytenoidectomy through an external approach. Some months ago I saw a patient with bilateral crico-arytenoid ankylosis, the clinical picture of which is much like the one discussed here. Due to marked hypertrophy of the false cords in this case it was impossible to reach the true cords by the intralaryngeal approach through an endoscope. I therefore decided to perform the arytenoidectomy through a laryngofissure. The thyroid cartilage was split in the midline, the hypertrophied ventricular folds were resected, and one arytenoid was removed without difficulty. Since the arytenoidectomy was performed in accord-

ance with Dr. Thornell's principles, I feel that this case offers further proof that the Thornell operation is a simple, feasible and desirable procedure.

DR. MAURICE F. SNITMAN: What does Dr. Thornell mean by the term adequate airway? Is it that amount which is sufficient to permit removal of the tracheotomy tube only, or does it imply that the patient has enough airway for physical stress? Can he explain the presence of a fairly good voice with sufficient glottic aperture during exertion?

About seven years ago Dr. Galloway advised the removal of the arytenoid cartilage, which procedure I have employed, and in order to obtain a good airway I have also removed some of the thyroarytenoideus muscle. Unfortunately the voice in all cases has been poor, but the patients have been capable of performing physical tasks.

DR. ROLAND RUSSELL: I should like to mention one point brought up by Dr. Thornell, which Dr. Holinger has already disagreed with.

I practice in a small town and see a good many cases of thyroidectomy. I have seen no case of cord paralysis in the hands of some men who have performed most of the operations. I do not believe that the inferior laryngeal nerve ever passes through the thyroid gland. If the right operation is done it is not necessary to cut the nerve. I think one should be charitable to the surgeon and not tell the patient that the inferior laryngeal nerve has been cut.

I agree with the desirability of routine laryngoscopic examination preoperatively. Several years ago a patient was referred to me because of voice change following thyroidectomy, and I found left cord paralysis. The surgeon said that the operation had consisted of removal of the right lobe of the thyroid, but upon reexamination I still found left cord paralysis.

DR. WILLIAM C. THORNELL, Cincinnati (closing): I wish to thank the discussors for their very nice comments. The first question was proposed by Dr. Pearlman. By tearing into the larynx I do not mean just a small opening or a small break in the mucous membrane, but maceration of the entire aryepiglottic fold and vocal cord; this could result if one were not familiar with endoscopic surgery. We have had two patients in which a small tear was produced through the mucous membrane in the region of the vocal process, and the patients still had a successful end result. Dr. Havens

has informed me that on several occasions he has made a small stab incision in the deepest portion of the wound extending into the subglottic area so as to afford better drainage inferiorly. I would like to emphasize that marked tearing and maceration of the posterior aspect of the vocal cord extending into the arytenoid area and interarytenoid space could produce a scar and prevent lateral displacement of the vocal cord.

Dr. Holinger mentioned the charitable remarks made by Haven (with which I agree) regarding the skill of the surgeon doing thyroid surgery. We have tried to be fair toward our general surgical colleagues. I have observed abductor vocal cord paralysis as a complication of thyroid surgery performed by some outstanding and skilled surgeons, and when such men encounter that complication I feel I should not be one to criticize.

Dr. Snitman asked whether an adequate airway could be obtained with such excellent voice as shown on the recording. Many of my patients have developed close approximation of the anterior third of the vocal cords with some bowing of the posterior two-thirds, so that on phonation there is tensing of the vocal cord and movement of the cord to the median line which results from contraction of the cricothyroid muscle which is supplied by the motor branch of the superior laryngeal nerve. This produces some approximation of the vocal cords and hence will help in the production of good voice. I have not had the opportunity to observe many patients who have had an external procedure carried out. I have tried this only once and was dissatisfied with the length of time and the difficulty of the procedure. As represented by the drawings, I think there is further separation anteriorly with the external approach, and closer approximation in our patients who had the intralaryngeal approach carried out. Dr. Snitman does not agree with this. I have noted in my patients some bowing of the cords so that on phonation the cords tend to move to the median line, while the glottic opening is large enough to produce an adequate airway.

There is one more point which I believe should be brought up, namely, how can we expect an adequate airway by simply removing the arytenoid cartilage? Simple removal of the space-consuming arytenoid cartilage will produce lateral displacement of the vocal cord. Secondly, the use of an electrocautery will also be a factor in producing further lateral displacement of the vocal cord. Third, freeing of the tension of the inter-arytenoid muscle may also be a factor insofar as this tension alone may hold the cords together in the median position. In the German literature there are reports

where the inter-arytenoid muscle has been removed producing some opening of the glottic chink. I have tried severing this muscle previous to intra-laryngeal arytenoidectomy, but was not too impressed with the lateral displacement of the vocal cords, so I then proceeded with removal of the cartilage.

## Therapeutic Approach to Sudden Deafness

JEROME HILGER, M.D.

MINNEAPOLIS, MINN.

(Summary)

Sudden deafness or, to broaden the definition, severe hearing impairment of sudden onset, is a symptom, not a disease entity. The known anatomic, histologic, pathologic and experimental data implicate vascular and water-balance mechanisms in the production of sudden deafness. Oft-quoted toxic or infectious sources have no substantiating background other than the obvious entity complicating infectious parotitis. As knowledge develops other sources may be identified, but at present sudden deafness must be considered principally under the headings of vascular obliteration; vascular dysfunction; or water imbalance in the cochlear segment of the labyrinth.

Vascular obliteration implies embolus, arteritis, thrombus or hemorrhage, and necessitates careful cardiovascular and blood evaluation in all cases to implicate the few. The majority of cases of sudden deafness result from vascular dysfunction or water imbalance due to endocrine dysfunction. The basal ganglia of the autonomic nervous system together comprise the focal center of autonomic function. Inherent or acquired dysfunction in this central ganglia area can express peripheral imbalance in the cochlea directly through neural routes, or indirectly through mediating untoward antigen-antibody reactivity, or humorally through the pituitary axis of the endocrine chain, as dysfunction in the thyroid, adrenals or gonads.

Representative cases of sudden deafness of these different types are presented and their therapy is discussed.

### DISCUSSION

DR. ROBERT H. EBERT (University of Chicago): Dr. Hilger has proposed a most interesting theory and one which deserves care-

ful consideration. I have only one major criticism. The anatomy, physiology and pathology of small blood vessels is not quite as simple as he has indicated diagramatically. He has presented the classical concept of vascular physiology. The terminal arteriole is assumed to lead directly into the capillary, and this simple endothelial tube connects with the collecting venule. Spasm of the terminal arteriole, according to this concept, interrupts the blood supply to capillaries and venules completely.

Direct observations by Chambers and Zweifach on living blood vessels have demonstrated that a more complicated anatomic and physiologic arrangement of small vessels exists. According to these authors, the terminal arteriole leads into a metarteriole which has a muscular wall with a single layer of muscle cells which are sparsely scattered at the end of the vessel. The metarteriole leads directly into the collecting venule without traversing any true capillaries. At intervals the metarteriole branches give off precapillaries which lead into the true capillaries. These then drain into collecting venules. At the junction of the precapillary with the metarteriole is a muscular sphincter. Blood may traverse either of these two routes in varying proportions and under different heads of pressure. It may go from arteriole to metarteriole directly into the collecting venule, a circuit in which the filtration pressure is relatively high, or it may go from metarteriole to precapillary to capillary to venule, a system in which the blood pressure falls more rapidly. A third route is often available, that is, a shunt directly from the main arteriole to venule.

In normal vessels there is a rhythmic change in tone and precapillary sphincters so that more blood is shunted through the metarteriole directly into the venule when precapillary sphincters are constricted and more through precapillaries and true capillaries when these channels are open. This phenomenon is called vasomotion.

The direction of filtration depends upon vasomotion. If a good head of pressure is maintained in the terminal arteriole, enhanced vasomotion tends to increase inward filtration, since the fall of hydrostatic pressure is greater in the true capillaries and the drainage from venules is good. Decreased vasomotion tends to promote outward filtration since hydrostatic pressure in the capillary bed falls, blood tends to collect in the venule, and increased back pressure favors an overall outward filtration. Vasomotion is controlled by both nervous and humoral factors and is a delicately balanced mechanism to regulate fluid and metabolite exchange in the tissues. It is possible, therefore, for certain types of edema to occur as a result of disturbances in the vasomotor mechanism.

If vascular spasm is the cause of sudden deafness, where is it occurring? Is it in the terminal arteriole, metarteriole or precapillary. These structures do not necessarily constrict at the same time and the end results are variable according to which structure is involved. I suspect that prolonged spasm of terminal arterioles sufficient to produce significant anoxia is exceedingly rate. I have seen it occur only in anaphylactic shock. It is possible, however, that local change in vasomotion might result in accumulation of edema fluid without changes in vascular permeability produced by anoxia.

I should like to show a motion picture of living vessels in the rabbit ear chamber to illustrate some of these points.

(Film)

I offer these remarks not as destructive criticism of Dr. Hilger's excellent paper, but only to suggest that the physiologic control of small blood vessels is excedingly complex, and that it is important not to over-simplify the concept of vascular spasm.

Dr. John R. Lindsay: I have greatly enjoyed this lucid exposition of the hypothesis of vascular spasm in relation to inner ear phenomena of various types. One point that should be brought to attention is that although there is a great dearth of information as to what happens to the inner ear in various diseases, gradual progress is being made in accumulation of fundamental information. For example, we lack information as to the pathologic changes in so-called toxic neuritis, yet it is common to have severe or profound deafness occur in association with some acute virus infections such as mumps. Experiments in recent years have shown that the sense organ may be damaged in various ways. We now have information that in streptomycin damage the main and primary injury is to the outer hair-cells. The outer hair-cells apparently get nourishment directly from the endolymph and not from the vessels. We know that localized damage to the sense organ is caused by acoustic trauma as well as severe blows to the head, and that the primary pathology consists in damage to hair-cells. More severe trauma causes loss of supporting cells in Corti's organ and degeneration of the peripheral cochlear neurone.

I mention these to call to your mind that we are accumulating fundamental information and that any hypothesis must take the established information into consderation to be acceptable. Such an hypothesis may be helpful if it gives a rational basis for treatment, yet we must continue the effort to get facts. We are indebted, as is otology in general, to Dr. Hilger for his discussion of this phenomenon tonight and over the last few years.

DR. S. L. SHAPIRO: I have considerable sympathy for Dr. Hilger's views because I have felt for a good many years that many of the internal ear troubles were of vasospastic origin, yet I think we would all agree that in the absence of direct proof his theory must remain an hypothesis. I have had occasion to use nicotinic acid for inner ear conditions and, occasionally, by injecting a good stiff dose in the office I have been able to abolish temporarily a spontaneous nystagmus. I have never been able to influence tinnitus with it or to obtain any brilliant results in cases of hearing impairment.

Unlike Dr. Hilger, I would hesitate to abolish completely the time-honored concept of neuritis, because we know that such a condition occurs or is considered to occur in other cranial nerves, notably the facial and recurrent laryngeal nerves where paralysis would be difficult to explain on a vasospastic basis.

One place where vascular spasm and the effect of medication upon blood vessels can be observed directly is the eye. A recent article in the American Journal of Ophthalmology spoke of a block by novocaine of the stellate ganglion, which produced marked dilatation in a short time of the retinal vascular tree and permitted the passage of an embolus of the central retinal artery, the entire process being observed through the ophthalmoscope. By inference, at least, this would prove that stellate ganglion block is one way of obtaining dilatation of cranial end-arteries, and I hope this method will be tried in cases of sudden deafness where vasospasm seems to be an important factor.

DR. JEROME A. HILGER (closing): Simplification and unification have a scaffolding purpose in the presentation of this material. In reality, the subject of sudden deafness is anything but simple. Obviously, there is more ignorance than knowledge of the basic fundamentals involved. One hypothesis stemming from the hypothalamus and operating through humoral and neural channels is supported by many fundamental facts and clinical observations. The gaps in our knowledge are enormous and do not admit of complete correction in the matter of therapy. Yet, enough is known of the basic changes in sudden deafness to give reasonable basis for a varied therapeutic approach and to make unreasonable a folded-hands, do-nothing attitude or a single stereotyped approach to all cases.

The correctness of an hypothesis is, in some respects, less important than the critical stimulus it may provide to others to deny its accuracy and formulate real fact.

# Abstracts of Current Articles

## EAR

**Vestibular Recruitment.**

*Arturo Tello, T.: Revista de Otorrlarng.* 11:31 (Aug.).

In performing the caloric test during a routine study of dizziness, the author has made observations which he interprets as evidence of "Vestibular Recruitment."

A preliminary report is made on twelve cases which fall into this group. They are classified as follows: Nine cases of excessive smokers, one of alcoholism, one due to streptomycin, and one of Bell's palsy.

His tests were performed by irrigating the external canals with 60 cc of water at 30° in 40 seconds, and repeated with water at 20.° Hypo-excitability and in some instances absence of any response is reported from irrigations at 30,° normal reactions and hyperactivity was observed when water at 20° was used. This phenomenon was most noticeable in the nine smokers.

Reasoning from the premise that nicotine has selective toxicity for the autonomic nervous system he associates hypo-excitability with excessive smoking.

In order to test this hypothesis he anesthetized the superior cervical sympathetic garglion on one side by injecting 1% novocaine and repeated the tests on the side of the injection he observed hypo-excitability resulting from irrigations at 30° and hyper-excitability with water at 20.°

HIGBEE.

**Malformations of the Auricle and the External Auditory Meatus: A critical Review.**

*Altmann, Franz: Archives of Otolaryngology* 54:115-139 (Aug.) 1951.

Typical of this author's fine, scholarly, well annotated and documented presentations is this carefully prepared critical review of malformations of the auricle and external auditory meatus. He has drawn on anthropology, embryology and genetics in his study. Conditions observed in ancient Egyptian mummies, in various races

and in animals as well as man are described. A bibliography, ten pages in extent, covers all of the available literature since 1930 and selected more important earlier publications.

In describing the malformations of this region, Altmann has divided his work into sections. Frequently he gives the distribution of the malformation in the population, its racial frequency, embryology, possible mode of transmission and a description of the common and some of the rarer types of malformation affecting each division of his subject.

Under the heading of the auricle are sections dealing with anomalies in position, variations in the relief of the auricle, true malformations, auricular appendages, congenital auricular fistulas, congenital arteriovenous fistulas and congenital tumors. The external auditory meatus is subdivided into changes in the shape of the meatus on the basis of individual and anthropologic variations and those due to true malformations, and congenital tumors. Attention is directed, under the first category of the latter heading, to the remarkable individual and racial differences in formation, especially of the width, of the external meatus and the author goes into detail in a discussion of the probable origin of hyperostoses of the outer parts, the exostoses of the innermost part of the osseous canal and the solitary osteomas. He relates the problem of ear exostoses to that of otosclerosis and adds some of his own speculations.

HILDING.

**Physical and Physiological Variables in Noise—Induced Hearing Loss.**

*Wheeler, Douglas: Archives of Otolaryngology* 54:267-272 (Sept.) 1951.

We have qualitative but inadequate quantitative information about noise-induced hearing loss and industry is now pointing up this inadequacy in its search for more quantitative information. Two factors are imperfectly understood in noise-induced hearing loss: (1) onset of loss, i.e., incidence of loss in a known population when the exposure variables are known and (2) rate of progression of loss, i.e., rapidly of increase in loss after it has been detected in persons under known exposure conditions.

Wheeler sets forth the following requirements to be met in collecting information: In brief form these may be stated to be, for the exposure variables—mean level of noise, frequency characteristics of noise and length of time the subject is exposed to the noise. For the subject variables—subjects should have normal hearing acuity

at inception of study, incipient ear disease and/or predisposition to hearing impairment should be ruled out by medical history and examination, young-adult age level is preferable, employment conditions should involve frequent exposure to high-level noises and labor turnover should be low.

The author undertook a study of 78 shipyard employees following the criteria set forth above; 43 men were rejected on the basis of audiometric acuity which was below normal and 35 men best fitting the requirements remained in the group for future study. Twenty-seven men were available for re-examination in six months. On the recheck there was slight improvement in the group hearing acuity at all frequencies except at 4000 and 8000 cycls. Caution is advised in interpreting these results since this represents only the first in a long series of tests and the trend will not be established without many additional measurements. Extrinsic elements possibly influencing these findings are postulated and the author lists several factors entering his study which must be borne in mind in evaluating the results. Illustrative audiograms are reproduced and information is presented in tabular form making it readily comprehensible.

HILDING.

## NOSE

**Purulent Meningitis from Obscure Sinus Infection.**

*Duclos, K., and Corcuerra, E.: Revista de Otorrlaryng.* 11:115-121 (Dec.) 1951.

Ten cases of purulent meningitis are reported as complications of sinus infection. The authors state that evidence of sinus infection is frequently not easily determined because the usual symptoms, and findings may not be apparent.

The examination is supplemented by x-ray findings whenever possible. The sinuses, particularly the maxillary, are punctured and irrigated. The return flow is examined cytologically. Infection of the sinuses may be of limited extent, which fact also contributes to the difficulty of detecting the lesions. Once established and defined a focus in any of the sinuses must be adequately treated by simple drainage, or extensive surgical exploration in conjunction with treatment of the meningitis.

HIGBEE.

## PHARYNX

**The Approach to the Nasopharynx.**

*Wilson, C. P.: Journal of Laryngology & Otology 65:738-744 (Oct.) 1951.*

The author points out the difficulty of diagnosis and treatment of the nasopharyngeal area, especially the surgical treatment. The problem is not new and the various approaches are discussed starting with Michaux in 1853 through the latest rhinotomy. The various nasal approaches are described as well as those through the face, mouth and palate.

Most approaches through the mouth and palate aim at antero-posterior incision of the soft and hard palates. They include partial to complete incision with varying areas exposed. The difficulty of handling flaps is noted.

The author conceived the idea of transverse incision between the hard and soft palate and then pushing the whole palate backwards. Extension of the incision downward to the outer side of the tonsillar fossa completes the procedure. The tendon of the tensor palatini muscle is divided and the whole soft palate displaced downward to the base of the tongue. Bleeding is negligible from the posterior branches of the palatine artery. The posterior spine can be removed to facilitate the whole procedure.

This affords a direct view of the posterior border of the septum, the posterior ends of the turbinates, the Rosenmuller fossa and eustachian tubes.

The wound is closed by three to five sutures to the mucosa of the hard palate. Postoperatively the patient can eat or talk and there is very little discomfort.

Diagrams and photographs are shown.

CUTLER.

**Poliomyelitis and Tonsillectomy.**

*Cohn, M.: Revista de Otorrlaryng. 11:39-47 (Aug.) 1951.*

The relationship between tonsillectomy and poliomyelitis is observed in a review of 560 cases of the disease collected during the last three years in the children's hospital of Santiago. In one case tonsillectomy was performed previous to the diagnosis.

The author reviews the literature briefly and makes an analysis of his own cases, with reference to the various theories which have

been advocated. He believes, with G. Anderson, that poliomyelitis is a disease which has largely infected the general population in a mild and undiagnosed form without paralysis, but nevertheless conferring a definite immunity.

In general he is in accord with the conclusions of Dr. Cunning in his survey conducted in the United States. The fact that bulbar poliomyelitis is twice as frequent in tonsillectomised patients prompts him to advocate deferment of all elective operations when poliomyelitis has reached epidemic proportions.

In the 560 cases of poliomyelitis studied there was one incident in which tonsillectomy preceded the diagnosis.

His conclusions are: 1. Tonsillectomies performed in the summer do not predispose to poliomyelitis. 2. A history of recent tonsillectomy in all cases of poliomyelitis is not a factor for consideration. 3. The urgency for tonsillectomies is much more important than the possibility of poliomyelitis.

HIGBEE.

**"Postoperative" Hyperrhinolalia (Rhinolalia Aperta).**

*Froeschels, Emil: Archives of Otolaryngology* 54:140-142 (Aug.) 1951.

Various means of recognizing hyperrhinolalia (audible passages of air through the nose at inappropriate times) are described. This presentation is concerned with the organic and functional insufficiency of the velum after adenoidectomy and/or tonsillectomy. It seems well established that true postoperative hyperrhinolalia exists but the author never has seen the development of "postoperative" hyperrhinolalia when he has examined the patient before operation without finding hyperrhinolalia. All of his patients showing strong hyperrhinolalia after surgery had at least slight findings before operation. Numerous cases of this speech trouble exist and some are recognizable by many people but the less evident cases require expert logopedic study.

A case of wrongly assumed postoperative hyperrhinolalia in a 4½ year old boy is presented. He was seen by the author only postoperatively but the history and findings pointed to a probable preoperative inefficiency of the velum in swallowing and an undetected hyperrhinolalia in the absence of examination by a speech expert. The hyperrhinolalia merely became more conspicuous after the adenoids, which acted like a damping cushion on air entering the nose, had been removed. Weakness of the velum was explained on the

basis of its always meeting a cushion of adenoids when trying to rise, resulting in functional "paralysis."

The author feels the point to be learned from this is that every candidate for adenectomy or tonsillectomy should be examined by a logopedist and, if insufficient velar function exists, the patient or his family can be warned of the presence of hyperrhinolalia and that it will be more conspicuous postoperatively. In the light of this finding in a patient, the otolaryngologist may wish to reconsider the situation.

HILDING.

## MISCELLANEOUS

**Cortisone in Behcet's Syndrome: Report on a Patient with Lesions of the Genitalia, Mouth, Pharynx and Larynx Necessitating Repeated Tracheostomies.**

*Kenet, David* S.: *Archives of Otolaryngology* 54:505-509 (Nov.) 1951.

The author brififlly reviews the history and characteristics of Behcet's syndrome and notes that up to the present time the treatment described in reports has been on an empirical basis. There was one reported instance of successful treatment with an antihistaminic drug but all other reports in the literature indicate no improvement or only slight improvement following any form of therapy. Because investigators found delay in the development of all elements of connective tissue in a rabbit's ear wound, the author felt it was of interest to determine the effects of Cortisone on the development of fibrosis at the sites of lesions, as well as on the rate of healing in Behcet's syndrome.

For 1½ years the author followed a 26 year old white male patient, at the Veterans Hospital, with an abortive form (having only two of the classic three signs) of Behcet's syndrome. This case was unusual in that the severity of the laryngeal lesions produced respiratory obstruction requiring repeated tracheostomies. It was felt that the patient had had a sufficient number of attacks with subsequent fibrosis to produce laryngeal stenosis. The edema, injection and ulcerations of the acute episodes added sufficient burden to an already inadequate airway to require surgical intervention and it was feared that repeated future attacks with fibrosis would necessitate a permanent tracheal stoma. In the latter part of the reported course of observation, Cortison therapy was instituted when the patient was re-admitted to the hospital with an acute episode of ulcerations. All

areas healed at the same rate as when no Cortisone therapy was given. In a 29 day period he received a total of 2.1 gm of Cortisone but no effects were noted on the onset and duration of symptoms of Behcet's syndrome.

HILDING.

**Pathology of Bell's Palsy.**

*Hall, Agnar: Archives of Otolaryngology* 54:475-477.

Investigators in this field have described Bell's palsy as being probably a pathologic entity chiefly characterized by a "dysregulation" of the circulation, probably taking place near the stylomastoid foramen, causing an ischemic paralysis. Lack of blood supply to the nerve leads to edema with subsequent degenerative changes. A vicious, but reversible, cycle is set up by the nerve being compressed in its bony canal thus impairing further the blood supply.

The author states that the present generally accepted therapeutic measure, when indications are correct, is decompression of the facial nerve from the stylomastoid foramen to the lateral semicircular canal. He reports that Kettel performed decompressions in 50 consecutive cases and concluded that the ischemia near the stylomastoid foramen, which is considered the cause of Bell's palsy, in most cases affects the nerve only as the most susceptible tissue but, in other cases, the surrounding, more resistant bone is also involved resulting in an ischemic bony necrosis. Flodgren published one case confirming Kettel's findings.

Three cases, in which resection of the mastoid cell and a decompression of the facial nerve were done and in which the findings at operation confirmed Kettel's investigations, are reported by the author.

HILDING.

# Books Received

**The Story of the Adaptation Syndrome.**

*By Hans Selye, M.D., Ph.D., Professor and Director of the Institut de Médecine et de Chirurgie expérimentales Université de Montréal.* Pp. 225 with illustrations. Montreal, Canada, Acta, Inc., 1952.

**Lung Abscess.**

*By R. C. Brock, M.S., F.R.C.S., F.A.C.S.*, pp. vi+197 with 60 figures. Springfield, Ill., Charles C. Thomas, 1952. (Price $7.75)

# Notices

## AMERICAN BOARD OF OTOLARYNGOLOGY

The American Board of Otolaryngology will conduct the following examinations:

October 6-10, 1952, in Chicago, Illinois, at the Palmer House.

April 21-25, 1953, in New Orleans, La., at Roosevelt Hotel.

DEAN M. LIERLE, M.D., Secy.
University of Iowa
Iowa City, Iowa

## HOME STUDY COURSE: ACADEMY

The 1952-1953 Home Study Courses in the basic sciences related to ophthalmology and otolaryngology, offered as a part of the educational program of the American Academy of Ophthalmology and Otolaryngology, will begin on September 1 and continue for a period of ten months. Detailed information and application forms can be secured from Dr. William L. Benedict, the executive secretary-treasurer of the Academy, 100 First Avenue Building, Rochester, Minnesota. Registrations should be completed before August 15.

## UNIVERSITY OF ILLINOIS

For the period of the National Emergency, the University of Illinois College of Medicine will offer a combined three year residency training program in Otolaryngology, which will include the basic course material, in fulfillment of Board requirements.

Residents will rotate through the Research and Educational Hospitals, the Illinois Eye and Ear Infirmary, the Hines Veterans Administration Hospital and the various affiliated institutions. The residency will be so flexible that should it be interrupted because of